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HERNIA OF THE LUNG

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TRUE hernia of the lung is a protrusion of the pleural-covered lung beyond its normal boundaries through an abnormal opening in the thoracic enclosure. Many of the earlier case reports were inaccurate because evisceration and prolapse were confused with true hernias. Hernias most commonly occur in the chest wall and are usually inside a sac of parietal pleura which may protrude into any layer of the thoracic parietes.

The rarity of hernia of the lung is proved by its infrequent mention in current literature and the small number of cases reported following major wars in which significant numbers of chest injuries have been recorded.

Roland[1] is given credit for describing the first case of hernia of the lung in 1499. By 1800, fifteen additional cases had been reported, but only nine of these, according to Montgomery,[2] were true pulmonary hernias. In the American Civil War[3] there were only three cases of hernia of the lung recorded. In the Crimean War[4] not a single case was reported. In the South African War (1899-1900) Makins,[5] in an extensive war experience, reported but one case, while Adams,[6] in the Russo-Japanese War, was able to find only five cases of pulmonary hernia among 20,000 wounds of the chest. In World War I[7] no cases of hernia of the lung were described. Montgomery and Lutz[8] were able to collect 165 cases from the literature up to 1925. Goodman,[9] in 1933, found 171 cases of hernia of the lung reported and added one case of his own. To date, less than 185 cases have been recorded in the medical literature.

CLASSIFICATION OF HERNIAS OF THE LUNG

Morel-Lavallée[10] was the first to undertake any basic work on pulmonary hernias and has outlined the simplest and most comprehensive classification.

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I. ACCORDING TO LOCATION
- A. Cervical
- B. Thoracic
- C. Diaphragmatic

II. ACCORDING TO ETIOLOGY
- A. Congenital
- B. Acquired
  - (1) Traumatic
  - (2) Consecutive
  - (3) Spontaneous
  - (4) Pathologic

According to Location

The cervical group are rare. The thoracic location is the most common and, to date, only one authenticated case of diaphragmatic hernia of the lung has been recorded in the world's literature.[11]

Cervical.—Cervical hernias usually occur through the superior aperture of the thorax in the interval between the sternocleidomastoid and scalenus anticus muscles following some tear or deficiency in Sibson's fascia which normally limits the cervical excursion of the dome of the pleura. Hernias in this location are most commonly congenital.

Thoracic.—The thorax is the most common location of hernias of the lung, probably because of the high incidence of trauma in this region. Another important factor is the anatomic weakness of the thorax in the interspaces anteriorly near the sternum and posteriorly near the vertebrae. The external intercostal muscles extend only to the costochondral junction anteriorly, and posteriorly the internal intercostal muscles extend only as far as the angles of the ribs. In these areas the intercostal muscles are representd by fibrous sheaths known as the anterior and posterior intercostal membranes. Most commonly, herniation occurs anteriorly because this congenitally weak area is covered only by the pectoral muscles which do not afford the support given by the trapezius, latissimus dorsi, and rhomboid muscles posteriorly.

Diaphragmatic.—The extreme rarity of the diaphragmatic hernia is probably due to the imbalance between the positive intra-abdominal and negative intrathoracic pressures, so that in injuries of the diaphragm, the abdominal viscera usually displace into the corresponding hemithorax while the lung collapses.

According to Etiology

Congenital.—Congenital hernias occur most commonly in the superior thoracic aperture or anteriorly in the interspaces near the sternum. According to Smith and Johnstone,[12] they are secondary to sternal clefts or deficiencies in the thorax which may result from arrested growth of structures composing the chest wall, or they may arise from defects covered by fibrous tissue which result from various intrauterine abnormalities such as amniotic bands, pressure of the elbow of a fetus against the chest wall, lack of amniotic fluid or pressure from uterine fibroids. If the lungs are in a state of collapse during uterine life, there is often no evidence of hernia until some time after birth. Hochsinger[13] claims that all lung hernias occurring in the first few weeks of life must be considered to be congenital in origin.

Acquired.—

Traumatic and Consecutive: The majority of hernias of the lung are acquired as a result of trauma. They have been reported secondary to operative procedures, following crushing and stab wounds of the chest, and as a result of perforating wounds due to rifle bullets and shell fragments. The proportionate rarity of hernia following penetrating wounds of the chest is due to the inherent elasticity of the lungs and concomitant pneumothorax or hemothorax in the majority of instances. Protrusions occurring some time after injury are referred to by Morel-Lavallée[10] as consecutive hernias. This term, however, is confusing and should be deleted from the classification because true hernias of the lung rarely, if ever, are apparent immediately after injury but appear later following healing of the wound.

Spontaneous: Spontaneous hernias occur as a result of local weakened resistance in the boundaries of the thoracic cavity along with an abnormal increase in the intrathoracic pressure, both of which must be present at the same time as neither alone seems to be able to account for a spontaneous hernia. Chronic bronchitis, whooping cough, straining at stool, heavy lifting, or anything which causes increased intrathoracic pressure are claimed to influence the herniation of lung through defects in the chest wall.

Pathologic: Hernias have been reported following abscesses of the chest wall and breast, empyema necessitatis, malignant tumors, and tuberculous caries of the ribs. To date, only one case of true hernia of the lung through the diaphragm has been recorded by Beale.[11]

INCIDENCE

In eighty-four cases collected by Auler[14] and Urbach,[15] the relative frequency of the various forms of pulmonary hernia was found to be as follows:

Congenital	20	(24%)
Traumatic	37	(44%)
Spontaneous	26	(31%)
Unknown	1	(1%)

A review by Montgomery and Lutz[8] of 165 cases, in seventy-nine of which the location was definitely mentioned, showed the following incidence:

Cervical	16	(20%)
Thoracic	62	(78.5%)
Diaphragmatic	1	(1.5%)
Congenital	14	(18%)
Traumatic	40	(50%)
Spontaneous	25	(32%)

SYMPTOMS

As a rule, there are few symptoms from pulmonary hernias. This is especially true in nontraumatic hernias. Usually the first complaint is that of tenderness over a new swelling in the chest wall which increases in size on forced expiration and disappears on inspiration. In the acute traumatic or

postoperative varieties, however, the picture is obscured by severe symptoms secondary to injury of the chest wall or pleura. Rarely, a pneumocele may be strangulated and will then present symptoms of local inflammation at the site of the strangulation. Several of the patients described in this report were without symptoms until an abnormal tumor was noticed on the chest wall.

DIAGNOSIS

The presence of a palpable orifice in the thorax through which a smooth, soft, crepitant, reducible hernia appears, the size and shape of which varies with respiration, should make the diagnosis evident. In the case of cervical pneumocele, however, the diagnosis may be more obscured. Incarceration of the herniated lung is unusual but does occur. Lillienthal[16] states that in true hernia only a small amount of the lung extrudes, and strangulation rarely occurs because the lung tissue is so easily compressible. According to all reports in the literature, the most important single diagnostic finding is the presence of a bulging mass increasing in size during expiration, reaching its maximum during forced expiration (Valsalva's maneuver), coughing, or muscular exertion, and diminishing in size during inspiration or quiet breathing. Quoting Olin,[17] according to Sauerbruch, if the reverse of this phenomenon occurs, namely, bulging on inspiration and retraction on expiration, a complication of diaphragmatic injury with prolapse of the abdominal viscera into the thorax and out through the chest wall may be suspected. In this connection it is important to emphasize that in our experience these diagnostic criteria, namely, the change in size of the thoracic bulge during the respiratory cycle and the Sauerbruch phenomenon have not been reliable. In four of the cases to be reported, it was impossible to determine preoperatively whether lung or one of the abdominal viscera occupied the hernial sac. Roentgenograms and fluoroscopic examinations of the chest may be of some diagnostic aid in this connection. In all four of our patients in whom abdominal viscera were in a hernial sac, the classical signs and symptoms already mentioned were present. It is important to note also that in all four of these patients the defect in the chest wall was in the vicinity of the ninth to the twelfth ribs and that in three of the patients the diaphragm had been injured, paralyzed by injury to the phrenic nerve, or surgically plicated prior to the time the patient presented himself for treatment of hernia of the chest wall. These considerations are of little practical importance, however, since regardless of location of the hernia or the contents of its sac, the technique of surgical repair remains the same.

In a differential diagnosis, the following conditions might be considered: subcutaneous emphysema, empyema necessitatis, lung suppuration and degenerating malignancy breaking through the chest wall, tumor of the thoracic wall, tuberculous caries of a rib, and gas bacillus infection of the thorax or chest wall with crepitation. According to Olin,[17] widespread subcutaneous emphysema may follow injury to the lung and pleura, and some of these subcutaneous air pockets may coalesce and form bulges in the chest wall which

yield crackling sounds when palpated. The air, however, in these accumulations is easily displaced and no changes occur during respiration as with pneumocele.

Gas bacillus infections[17] may occur in crushing injuries of the chest wall and cause a crepitant bulge in the soft tissues. However, signs of infection characteristic of this condition may be recognized, and the phenomena elicited during the Valsalva test are not characteristic of hernia of the lung.

TREATMENT

True hernias of the lung seldom heal spontaneously and are rarely fatal.

Palliative measures such as local compression of the hernia by pads, corsets, and elastic apparatuses may relieve symptoms but are usually not curative. The treatment is essentially surgical.

Vogler,[18] in 1898, suggested a plastic operation using a bone flap from the sternum. Vulpius[19] in the same year described a procedure in which strips of ribs were crossed and sutured in place with silver wire. The hernia repaired in this manner recurred. Tuffier[20] produced a cure by merely freeing and closing the sac. Graham,[21] in 1922, packed the sac of a cervical pneumocele with iodoform gauze and obtained a permanent cure. Goodman,[9] in 1933, excised a hernial sac and exacted a cure by covering the defect with periosteal flaps developed from the ribs above and below the margins of the hernial orifice. Winkel,[22] in 1935, employed fascia lata in one case. Keller[23] also used fascia lata during World War I for the repair of chest wall defects. He is now convinced that utilization of the elements of the chest wall (rib, periosteum, and muscles) results in a better repair. Gadzhiev,[24] in 1936, utilized two muscular periosteal osseus flaps with pedicles developed by splitting lengthwise the ribs above and below the hernial sac and sutured the flaps centrally over the defect.

A tantalum plate has been used by Paulson[25] to cover a chest wall defect with an associated hernia of the lung. However, he has abandoned this method of treatment because of the difficulty encountered in immobilizing the metal plate on a constantly moving structure such as the thorax.

It is safe to conclude that there has not been, in the past, sufficient experience in the surgical treatment of hernias of the chest wall to establish any one acceptable surgical technique. Experiences in repairing large defects in the chest wall in eleven patients, seven of whom had defects associated with true hernias of the lung, justifies the following conclusions:

1. Any defect of the chest wall, regardless of size, can usually be repaired by plastic procedures involving only structures which are part of the chest wall, namely, rib, periosteum, and muscle.

2. The most important feature in the repair of pulmonary hernias is the covering of the defect with sturdy bone or with periosteum which will produce bone. Ribs, after division and release from their periosteal "envelopes," can be shifted to bridge the defect and anchored in the desired position by pericostal

fixation, by suturing to fascia or adjoining periosteum, or by fastening rib to adjacent rib with absorbable sutures threaded through drill holes. The most satisfactory repair of average-sized defects is obtained by the suturing of periosteal flaps developed from ribs immediately above and below the margins of the hernial orifice.

3. The freeing of adhesions and of the lung about the margins of the hernial orifice combined with the creation of a local pneumothorax is important in producing a "cushion" between the lung and freshly repaired chest wall. This reduces to a minimum the possibility of the lung's being forced into the chest wall by coughing and straining during the period when the repair is weakest.

4. Many defects of the chest wall associated with hernia of the lung will in the course of time become increasingly rigid, but the hernia, although smaller, will persist.

5. The endotracheal administration of the anesthetic agent is necessary when surgical repair of defects of the chest wall and hernia of the lung is attempted. This insures safety when the pleura is opened widely and also makes it possible to leave the lung in the desired degree of inflation.

The criteria for the selection of patients for the surgical repair of hernia of the lung and the principles of the operation which we have employed are demonstrated by cases 1 to 11.

CASE REPORTS

CASE 1.—On Dec. 3, 1943, a 35-year-old white soldier was admitted to a station hospital with a history of having fallen on an obstacle course eight days preceding admission. There was no history of pain at the time of injury and the soldier recalled no direct trauma to the chest. However, on the evening of the accident, he noticed, for the first time, pain localized in the left chest following coughing. Within four days following the accident, a small lump made its appearance beneath the skin in the left chest on coughing or sneezing. The past history revealed that twelve years preceding this admission, while playing football, the soldier fell on another player's heel, sustaining a contusion of the left chest anteriorly. The chest was moderately sore and the patient stated that he felt a hole in his chest with his fingers, and that he noticed a protrusion of the chest which appeared on coughing, similar in character to the one described at the time of his admission to the station hospital. On admission to the station hospital, there was described a soft herniation of the lung through the interspace between the eighth and ninth ribs in the left anterior axillary line. The defect admitted one finger easily. The remainder of the physical examination was not remarkable.

The soldier was transferred to Walter Reed General Hospital Jan. 18, 1944, where examination revealed the defect in the left chest wall at the ninth interspace in the left anterior axillary line with bulging of the lung through the orifice when the patient held his nose and mouth and forcibly expired (Fig. 1). The defect measured 3 by 4 cm. in diameter. Special roentgenograms of the chest on inspiration and expiration Jan. 28, 1944, showed herniation of the lung through the interspace between the ninth and tenth ribs on the left side anterolaterally (Fig. 2). Herniation occurred only during forced expiration with a closed glottis.

Operation.—On Feb. 1, 1944, under endotracheal oxygen-ether anesthesia, an incision was made over the ninth interspace, left chest. Immediately after incising the skin, the pleura was entered. Closer examination showed congenital absence of the intercostal bundles.

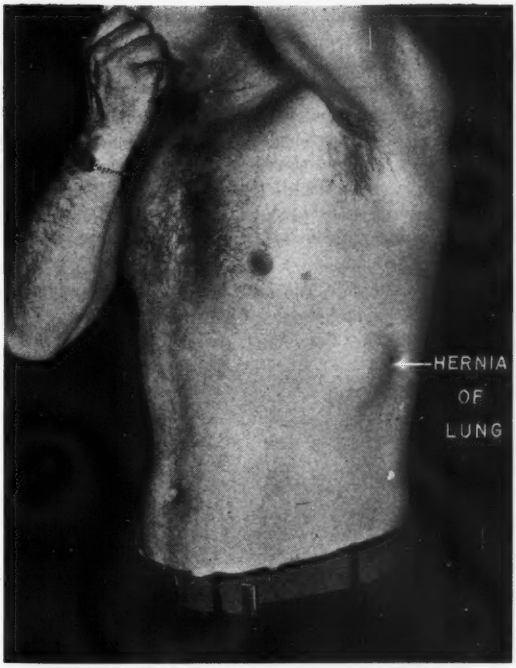


Fig. 1 (Case 1).—Herniation of lung through the ninth interspace, left, produced by performing Valsalva's maneuver.

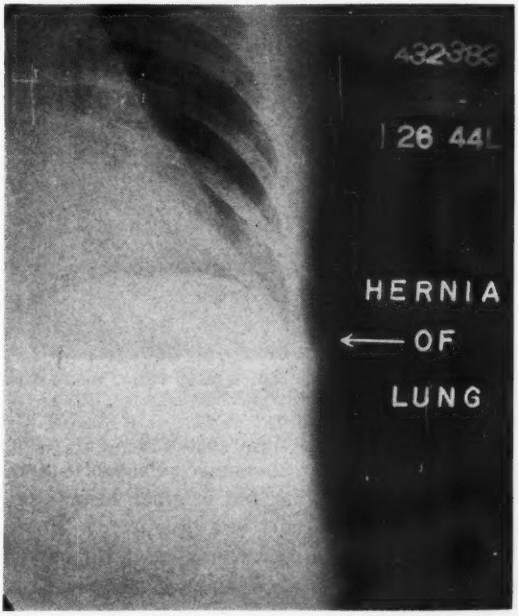


Fig. 2 (Case 1).—Photograph of roentgenogram showing air containing herniated lung outside of chest wall, beneath skin. Roentgenogram taken during forced expiration.

The adherent lung was freed from the margins of the hernial orifice and was not completely expanded while the chest was being closed, deliberately creating a localized pneumothorax pocket to prevent the lung from coming in contact with the freshly repaired wound during the early interval following the operation. The pleura was closed with interrupted fine silk sutures. Periosteal flaps were developed inferiorly from the anterior surface of the ninth rib and superiorly from the tenth rib and sutured over the defect in such a manner that the gap between the ribs was bridged by a complete layer of periosteum. The subcutaneous tissue and skin were approximated with fine silk sutures and a pressure dressing was applied.

The wound healed per primam. The patient completed a reconditioning program and re-examination May 10, 1944, four months following operation, showed no evidence of recurrence of the hernia (Fig. 3). The soldier at this time was without symptoms.

Fig. 3 (Case 1).—Postoperative scar. Photograph taken during forced expiration demonstrates absence of pneumocele.

Comment.—This patient undoubtedly originally had a defect of the chest wall with an associated hernia of the lung. Probably there was increasing rigidity of the chest wall following the original injury so that the defect became less noticeable and the hernia less prominent. Following the second injury, the patient was again aware of a bulging of the chest wall in the same vicinity. Surgical treatment, by developing periosteal flaps to bridge the defect, resulted in a cure.

CASE 2.—A 21-year-old white soldier sustained a penetrating shell fragment wound of the left thorax and upper abdomen. Exploration of the thoracic wound revealed a laceration of the lower lobe of the left lung which was of such severity that partial lobectomy was performed. Also, at the same time a metallic foreign body, which had lacerated the diaphragm and traversed the transverse colon, was removed from the left pleural cavity. The diaphragm was repaired through a transthoracic approach and a colostomy was performed proximal to the site of perforation. During the course of evacuation, an empyema, left, was treated by rib resection and open tube thoracostomy. On June 11, 1944, the colostomy in the right iliac fossa was closed and the soldier was returned to the Zone of the Interior.

At the time of admission to Walter Reed General Hospital, a drainage tube was still in place in the chest and a roentgenogram Sept. 18, 1944, showed no apparent empyema cavity. The tube was removed and a roentgenogram Oct. 25, 1944, showed the chest to be entirely normal. On Feb. 28, 1945, a thoracostomy, left, was performed for recurrent empyema, and by April 17, 1945, the tube had been removed and the wound was completely healed. About July 4, 1945, weakness of the left anterolateral chest wall was for the first time observed, and a definite defect could be palpated at the level of the fifth and sixth ribs in the anterior axillary line. The defect admitted two and one-half fingers. A bulge the size of the defect appeared on coughing or forced expiration, and a hernia of the lung was evident. The patient was not willing to have an operation because the symptoms caused by the hernia were not troublesome.

Comment.—Unfortunately, this patient was not operated upon. However, it is safe to conclude that this case represents a true hernia of the lung through a defect in the chest wall secondary to trauma.

CASE 3.—A 24-year-old white soldier sustained a penetrating bullet wound of the right chest July 7, 1944. The point of entrance was in the right anterior chest, third interspace; the point of exit, right midaxillary line, fifth interspace. There was hemoptysis for from two to three days following injury. The patient received first aid aboard a destroyer and was then removed to a general hospital, where, July 21, 1944, roentgenograms of the chest revealed a mass in the left lung field. Further examination disclosed what was thought to be a tumor mass in the left lung at the level of the left second interspace, just behind the sternum. There was no history of weight loss. Biopsy of a right inguinal node, Aug. 5, 1944, was reported as chronic inflammatory tissue with hyperplasia.

On admission to Walter Reed General Hospital the physical examination was not remarkable except for a scar at the third anterior interspace, right, representing the point of entrance of the missile, and a transverse scar, 8 cm. in length, in the right midaxillary line at the level of the nipple, representing the wound of exit. A previous diagnosis of allergic bronchitis was confirmed by a consulting allergist and the patient was placed on hyposensitization therapy. He was observed for thirty days and check roentgenograms revealed no change in the size or appearance of the tumor mass in the chest during this interval. A diagnosis of osteochondroma, left anterior chest wall, was made.

Operation.—On Jan. 15, 1945, under endotracheal ether anesthesia, an incision was made between the third and fourth cartilages in the anterior left thoracic wall. The incision extended from the sternum laterally for a distance of 20 cm. The pectoral muscles were divided and the intercostal space between the third and fourth costal cartilages exposed. The tumor could be palpated near the intercostal muscles. It was evident, both from roentgenographic findings and from clinical examination, that most of the tumor was intrapleural. Accordingly, the intercostal muscle over the tumor was incised and a mass 4 by 4 cm. in diameter was found attached to the third costal cartilage. It was easy to free the tumor by fracturing it off the cartilaginous attachments with a periosteal elevator. The point of fracture exposed what seemed to be normal cartilage. The tumor grossly

appeared benign and the cartilage was not resected. After removal of the tumor, the lung was inflated and the defect of the chest wall was closed by suturing what remained of the intercostal muscles, between the two cartilages, with interrupted No. 2 silk sutures. The pectoralis minor and major muscles were approximated with interrupted fine silk sutures. The subcutaneous tissue and skin were approximated with interrupted fine silk sutures, and a dry sterile compression dressing was applied.

The immediate postoperative course was uneventful and the wound healed per primam. The soldier went on a twenty-one day convalescent furlough Feb. 21, 1945, returning March 16, 1945, at which time he complained only of asthma. By March 21, 1945, however, while awaiting disposition, a hernia of the lung was first noticed at the recent operative site. A definite defect 6 by 8 cm. in diameter was palpable, and a large mass of lung presented itself as a crepitant bulge through the margins of the defect.

Operation.—On March 28, 1945, under endotracheal ether anesthesia, the scar of the former incision in the left anterior chest wall was excised and the underlying muscles divided. There was a large defect between the third and fourth costal cartilages through which a portion of the left upper lobe herniated. The defect measured 7 cm. in length and the distance between the two cartilages was 6 cm. A sac of parietal pleura 8 by 10 cm. in diameter, containing lung, had protruded beneath the pectoral muscles and was firmly attached to these structures, so that during periods of straining a portion of the left upper lobe was forced out and into the chest wall beneath the pectoral muscles. The adherent portion of the lung was dissected free and allowed to drop back into the pleural cavity. The pleura which had herniated out over the ribs was likewise freed and allowed to fall into the thorax. The cartilage of the fourth rib was freed back to the costochondral junction, just beyond which the fourth rib was fractured. The third costal cartilage and rib were approximated as close as possible to the fourth fractured rib by pericostal and perichondral sutures of No. 1 chromic catgut. The muscles of the chest wall were then approximated over the chest defect in layers with medium silk. Interrupted fine silk sutures were placed in the pectoral major, subcutaneous tissue, and skin. An attempt was made only to inflate the underlying lung partially, creating a deliberate residual pneumothorax to act as a "cushion" between the lung and freshly repaired chest wall during the early period of postoperative coughing or straining. Fifty thousand units of penicillin were placed in the pleural space at the end of the operation. The wound healed per primam and there was no evidence of recurrence of hernia at the time of re-examination May 17, 1945, almost two months following operation.

Comment.—This is a case of proved true hernia of the lung with extension of the pleural sac out into the chest wall and up beneath the pectoralis muscles so that on forced breathing or straining a portion of the upper lobe of the left lung actually presented in the pleural pocket between the chest wall and the pectoralis muscles. A cure was effected by the simple procedure of freeing the lung from the margins of the defect and then covering the defect with rib and cartilage which were shifted into the desired positions and fixed with pericostal sutures.

CASE 4.—A 25-year-old soldier sustained a penetrating shell fragment wound of the right chest Jan. 15, 1945. Emergency treatment was administered at an evacuation hospital, at which time the wounds were closed by suture. Intercostal tube drainage with water seal was carried out for three or four days, following which aspiration of the right chest was performed on three occasions.

At the time of admission to Walter Reed General Hospital, the patient complained only of mild pain in the right chest without hemoptysis or cough. There was a linear scar, well healed, 11 cm. in length, in the right anterolateral chest. Another well-healed scar, 9 cm. in length, appeared along the vertebral border of the right scapula. The lungs were

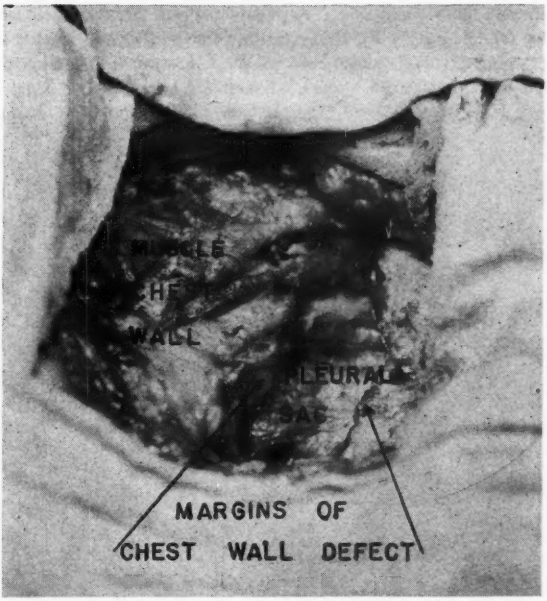


Fig. 4 (Case 4).—First stage of operation. The muscles of the chest wall have been divided, disclosing the margins of the defect, adherent lung, and pleural sac.

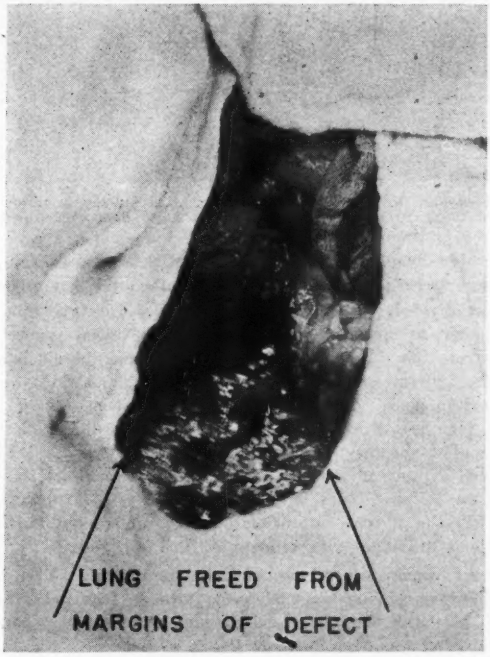


Fig. 5 (Case 4).—Second phase of operation. The lung and pleural sac have been freed from the margins of the defect.

normal to auscultation and percussion. A roentgenogram of the chest March 15, 1945, revealed a metallic foreign body 7 by 7 mm. in diameter and several smaller metallic foreign bodies in the right lung. There was absence of 8 cm. of the right sixth rib laterally. The patient complained of pain over the defect. Examination revealed a definite defect in the chest wall in this area which admitted three fingers for a distance of 1 cm. On coughing or forced expiration, with the glottis closed, a definite bulge appeared through the defect.

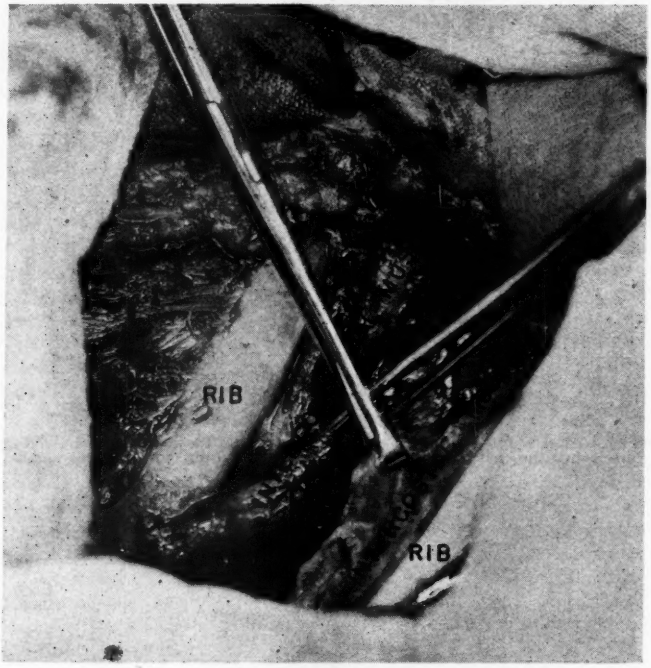


Fig. 6 (Case 4).—Third step in operation. The intercostal muscle bundles, to be used as the first layer in closure of the defect, have been developed.

Operation.—On June 8, 1945, under endotracheal ether anesthesia, an incision was made over the defect in the right anterolateral chest wall at the level of the sixth rib. The skin and muscles were divided. On dissecting the muscles free, the lung was exposed and was found to be adherent to the chest wall (Fig. 4). The adhesions were divided and the lung was separated from the chest wall over an area 10 cm. in diameter (Fig. 5). The periosteum of the fifth and seventh ribs was developed into flaps which were made to bridge the defect and sutured in place. The first layer of closure consisted of intercostal muscles which were brought together with interrupted sutures of medium silk (Figs. 6 and 7). The ribs were then pulled together with retractors and the two flaps of periosteum were joined with interrupted sutures of medium silk (Fig. 8). No effort was made to reinflate the lung fully because it was desirable to maintain a residual localized pneumothorax. The muscles of the chest wall were approximated with interrupted fine silk sutures. The skin was closed, without drainage, with interrupted fine silk sutures.

The postoperative convalescence was entirely uneventful. The wound healed per primam and the soldier was sent on a convalescent furlough four weeks after his operation. Re-examination Aug. 14, 1945, showed no evidence of recurrence of hernia, and the area of defect recently repaired was firm.

Comment.—This patient had a true hernia of the lung through a defect in the chest wall which was successfully repaired by suturing periosteal flaps developed from the ribs above and below the margins of the defect.

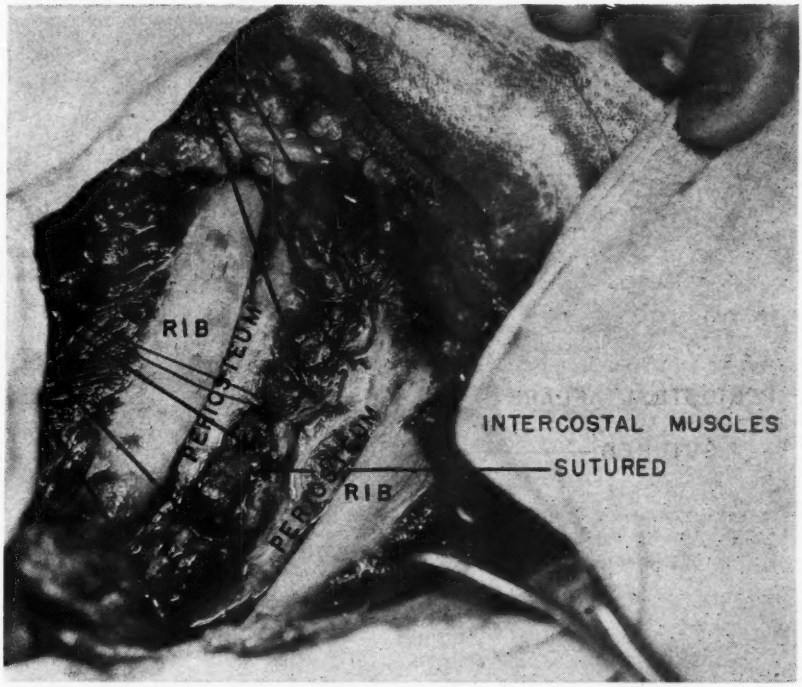


Fig. 7 (Case 4).—The intercostal muscle bundles have been approximated with medium silk sutures. Periosteal flaps have been developed from the ribs above and below the margins of the defect.

CASE 5.—On Dec. 27, 1944, a 31-year-old soldier sustained a penetrating rifle bullet wound of the right chest. At a field hospital thoracotomy with exploration of the right chest was performed. The chest was closed with intercostal tube-water seal drainage for ten days.

On arrival at Walter Reed General Hospital March 23, 1945, the patient complained only of pain in the right chest. On physical examination there was flattening of the right anterior chest under a scar above the right nipple. The lungs were clear to auscultation and percussion and the heart was normal. While under observation, the soldier continued to complain of pain in the right anterolateral chest and following return from furlough May 8, 1945, a definite bulge was noted for the first time, anteriorly, at the level of the fifth and sixth ribs. On forced expiration and coughing, a definite bulge appeared in the fifth interspace and disappeared on quiet breathing and inspiration. The defect in the chest wall measured 4 by 5 cm. in diameter. Surgical repair was offered as a method of treatment to the patient but was refused.

Comment.—Because of the position of the defect at the level of the fifth interspace in the right anterolateral chest wall, it can be assumed that this

patient had a true hernia of the lung. If operation had been accepted, it was anticipated that repair of the defect could have been accomplished by developing periosteal flaps from the ribs above and below the margins of the hernial orifice and by approximating these flaps over the central portion of the defect.

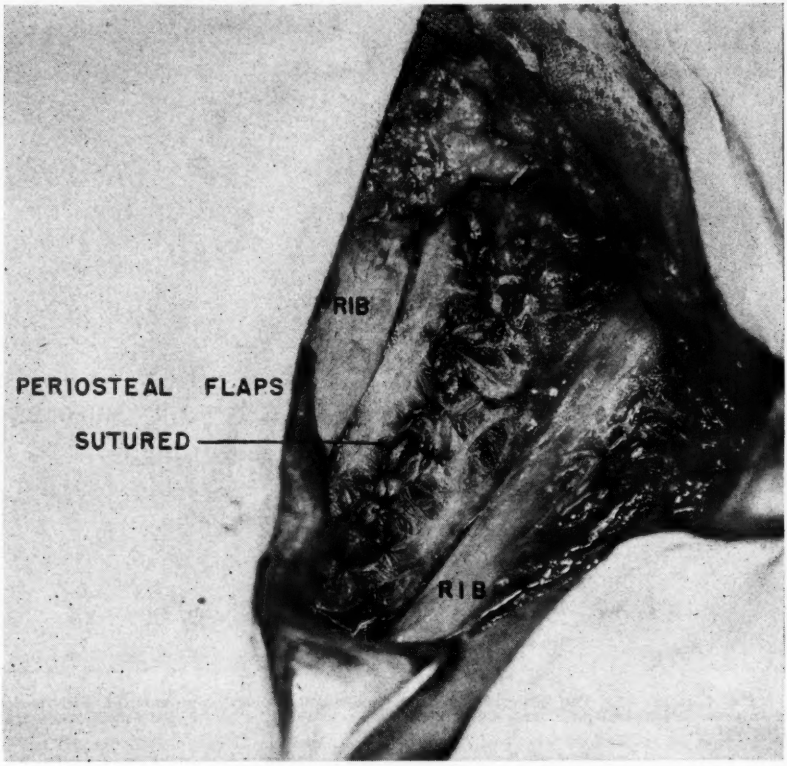


Fig. 8 (Case 4).—Final stage of operation. Periosteal flaps developed from ribs bounding the defect have been sutured over the intercostal muscles, forming a second layer of closure.

CASE 6.—A 21-year-old soldier sustained a shell fragment wound of the right chest and right arm Dec. 26, 1944. At a field hospital the wounds were débrided and resection of a portion of the right third rib was performed. The resulting defect was so large that it was necessary to use the pectoral muscles to close the wound. Intercostal catheter-water seal drainage was instituted for several days. On arrival at Walter Reed General Hospital April 7, 1945, physical examination was negative except for flattening of the right pectoral region and impaired percussion note and distant breath sounds over the posterolateral chest on the right. At the site of the previous resection of the third rib, there was a definite crepitant bulge, 3 by 3 cm., which appeared when the patient forcibly expired with the glottis closed. Because of the relatively small size of the defect, surgical repair was postponed, and during the subsequent course of convalescence at Walter Reed General Hospital, there was gradual but very definite increasing rigidity of the chest wall over the area of the defect. Re-examination July 16, 1945, showed complete absence of a palpable defect and no evidence of hernia of the lung.

Comment.—This case demonstrates that there is usually progressive increasing rigidity of the chest wall over a defect following surgical repair. In this patient, according to the history, the defect had been so large that the pectoral muscles were used in closure. Because of the position of the defect, it is fair to conclude that the contents of the hernia sac was lung. This is probably an example of a traumatic type of pneumocele.

CASE 7.—A 34-year-old soldier sustained a penetrating machine gun bullet wound of the left chest Feb. 12, 1945. The point of entrance was in the left anterior chest at the level of the third and fourth ribs; there was no point of exit. At a field hospital the wounds were débrided, portions of the third and fourth ribs were resected, and a laceration of the lung was repaired.

On admission to Walter Reed General Hospital May 2, 1945, there was a healed scar 11 cm. in length in the left pectoral region. There was a defect in the chest wall immediately beneath the scar measuring 8 by 6 cm. in diameter. On forced expiration, coughing, or straining, the lung herniated through the defect and presented two inches above the surface of the chest wall.

Operation.—On June 13, 1945, under endotracheal oxygen-ether anesthesia, an incision 15 cm. in length was made over the midportion of the defect in the left anterior chest wall between the second and fifth costal cartilages and ribs. The pectoral muscles were incised, exposing the pleura and lung. The lung and attached pleura were freed from the chest wall and an extrapleural pneumothorax pocket deliberately created. This space extended well away from the margins of the wound. The periosteum of what remained of the third and fourth ribs was stripped from bone, and the flaps were sutured with intercostal muscle over the defect. The fifth rib was fractured just lateral to the costochondral junction. The fifth cartilage was pulled upward and held in place over the defect with perichondral sutures of No. 1 chromic catgut. The muscles of the chest wall were approximated in layers with interrupted fine silk sutures. The skin was closed without drainage.

Examination of the patient Aug. 7, 1945, two months following operation, showed the left anterior chest wall to be rigid. There was no evidence of recurrence of the hernia and the patient was without symptoms.

Comment.—This patient had a traumatic pneumocele. A possible explanation of the persistence of the defect in the chest wall is the partial removal, either traumatic or surgical, of the periosteum of the third and fourth ribs at the time of injury or operation. A satisfactory repair and cure were obtained by combining the use of the residual periosteal flaps and perichondral sutures about a displaced rib.

In the following four cases are reported the classical signs and symptoms of defect of the chest wall with associated hernia of the lung according to the usual criteria. At the time of operation, no hernia of the lung was encountered, but defect of the chest wall was repaired in the usual manner and cure obtained.

CASE 8.—A 19-year-old soldier was admitted to Walter Reed General Hospital Nov. 30, 1944, with a history of having sustained a penetrating rifle bullet wound of the right chest July 12, 1944, with resulting fractures of the eighth, ninth, and tenth ribs, right, and hemothorax, right. A large defect 10 by 6 cm. in diameter with loss of part of the eighth, ninth, and tenth ribs was described in the overseas record, and repair of the defect was carried out by suturing the diaphragm to the edge of the wound and then closing the pleura.

On admission to Walter Reed General Hospital the wound in the anterolateral aspect of the right chest was healed. There was a bulge in the middle portion of the wound on Valsalva's maneuver. There was a defect in this area measuring 6 by 4 cm. in diameter. It was decided to observe the lesion for an interval, during which time it was hoped that the chest wall would become rigid. The patient was given a twenty-one day convalescent furlough, and on his return to the hospital surgical repair of the hernia was performed because of continued complaints of pain and persistent bulge in the right anterolateral chest wall.

Operation.—On March 2, 1945, under endotracheal ether anesthesia, the scar of the previous operation was excised. The bony thorax was exposed and the defect, 4 by 6 cm. in diameter, located. The soft tissue bulge in the midportion of the defect was opened and the peritoneal cavity entered. The liver presented in the wound. The peritoneum was closed with interrupted sutures of fine silk. The periosteum of the ribs above and below the defect were stripped to form flaps which were then sutured over the defect. The muscles of the chest wall were approximated with interrupted sutures of fine silk, and the skin was closed without drainage.

The wound healed per primam. The patient was sent on a convalescent furlough. Re-examination May 23, 1945, showed a good repair of the defect in the anterolateral chest wall with no evidence of residual or recurrent hernia.

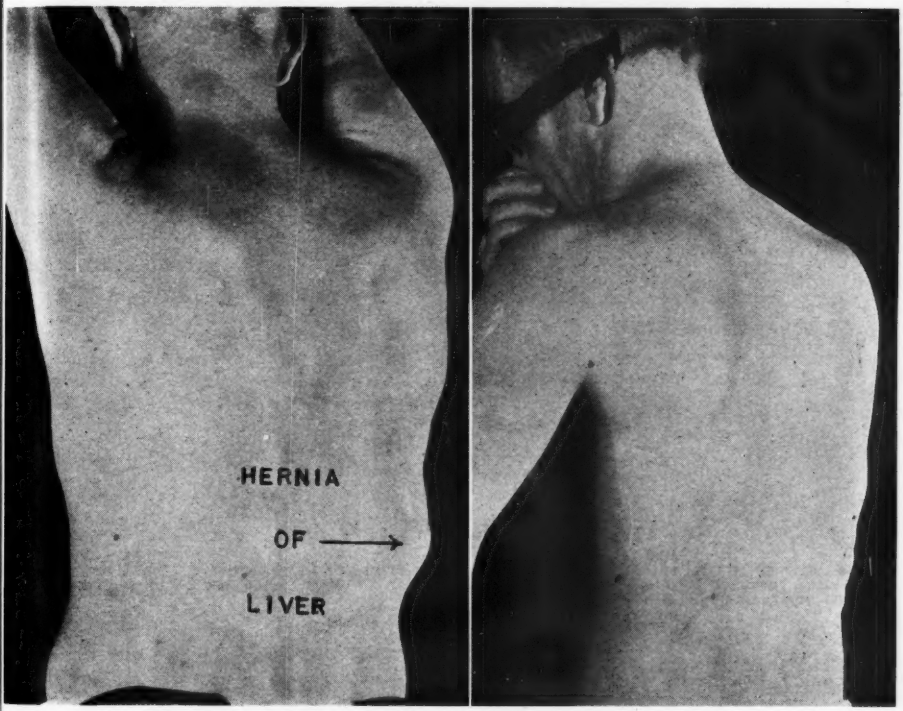
CASE 9.—On Sept. 27, 1944, a 26-year-old soldier sustained a perforating wound of the right chest, lung, diaphragm, and liver, with fracture, compound, comminuted, sixth and seventh ribs on the right, and hemothorax. At a general hospital repair of the diaphragm, liver and lung was carried out and later an empyema and subphrenic abscess on the right were drained.

On admission to Walter Reed General Hospital, all wounds were healed. Roentgenogram of the chest was normal except for moderate thickening of the pleura in the area of the right ninth rib. The patient complained only of pain in the right chest over an area of defect in the right ninth and tenth ribs which measured 5 by 9 cm. in diameter. Four fingers could be inserted into the defect. During Valsalva's maneuver, there was a small, soft, tender bulge through the defect. During a period of observation which followed, the soldier continued to complain of pain over the area of the defect. However, it was thought inadvisable to attempt surgical repair because the chest wall was becoming more rigid. In the meantime, because of very persistent pain, limited to the anterolateral lower portions of the chest, right, phrenemphraxis was performed May 9, 1945, with immediate subjective relief. Eventually, because of the recurrence of symptoms of discomfort over the bulge between the right ninth and tenth ribs, repair of the hernia was decided upon.

Operation.—On June 21, 1945, under endotracheal oxygen-ether anesthesia, an incision was made immediately over the defect approximately at the level of the ninth rib. Upon dividing the muscles, the peritoneal cavity was entered and the liver exposed. The tissues over the liver were dissected free and the diaphragm was found to be extremely high because of the previous surgical paresis of the right phrenic nerve. The peritoneum and edge of the diaphragm were approximated in layers with interrupted medium silk sutures. The rib above the defect was then freed of periosteum and its anterior end fractured. Four encircling sutures of No. 1 chromic catgut and one of No. 2 chromic catgut were placed about the rib, the end of which was pulled as far down as possible to bridge the defect and was then sutured to the adjoining muscles and fascia. Encircling sutures were placed in a similar manner about the rib, marking the inferior margin of the defect. It was then pulled superiorly and anchored in place by suturing the encircling pericostal sutures into the adjacent muscles and periosteum. The periosteum from both ribs was turned back in the form of flaps and sutured in place. The muscles of the chest wall were approximated in layers with medium silk. The skin was closed with fine silk without drainage.

During the immediate postoperative period, the patient complained of some discomfort and of motion in the chest wall at the site of operation. However, at the time of his final examination Aug. 1, 1945, the chest wall was rigid and there was no evidence of hernia.

CASE 10.—A 27-year-old soldier sustained a penetrating rifle bullet wound of the right chest Sept. 29, 1944. The point of entrance was in the eighth interspace to the right of the midclavicular line; the point of exit was in the tenth right interspace, posteriorly, 3 cm. to the right of the spinous processes. At a field hospital, a right posterolateral thoracotomy with resection of portions of the right ninth and tenth ribs was performed Sept. 29, 1944. Lacerations of the lower lobe of the lung, right diaphragm, and liver were repaired. Penicillin was instilled in the pleural space and water-seal intercostal drainage was instituted for several days.



A. *B.*

Fig. 9 (Case 10).—*A*, Preoperative view of bulge in right chest wall during forced expiration (Valsalva). *B*, Photograph of patient following operation. Note incision, right posterolateral chest, and absence of hernia. Figs. 10 to 14 illustrate the technique employed in this case.

On admission to Walter Reed General Hospital the only significant finding was the absence of 10 cm. lengths of the ninth and tenth ribs in the right posterior axillary line, resulting in a defect which admitted five fingers a distance of about 4 cm. During forced expiration (Valsalva), a soft, moderately tender bulge appeared through the defect (Fig. 9, *A*). This bulge measured 12 by 6 by 8 cm. in diameter at the height of forced expiration. On quiet breathing and inspiration, the mass disappeared. According to the test devised by Sauerbruch, there was no associated injury of the right leaf of the diaphragm. It was impossible to determine preoperatively the exact nature of the contents of the hernia.

Operation.—On Aug. 1, 1945, under endotracheal oxygen-ether anesthesia, the old scar over the right posterolateral chest wall was excised. Incision of the soft tissue in the central

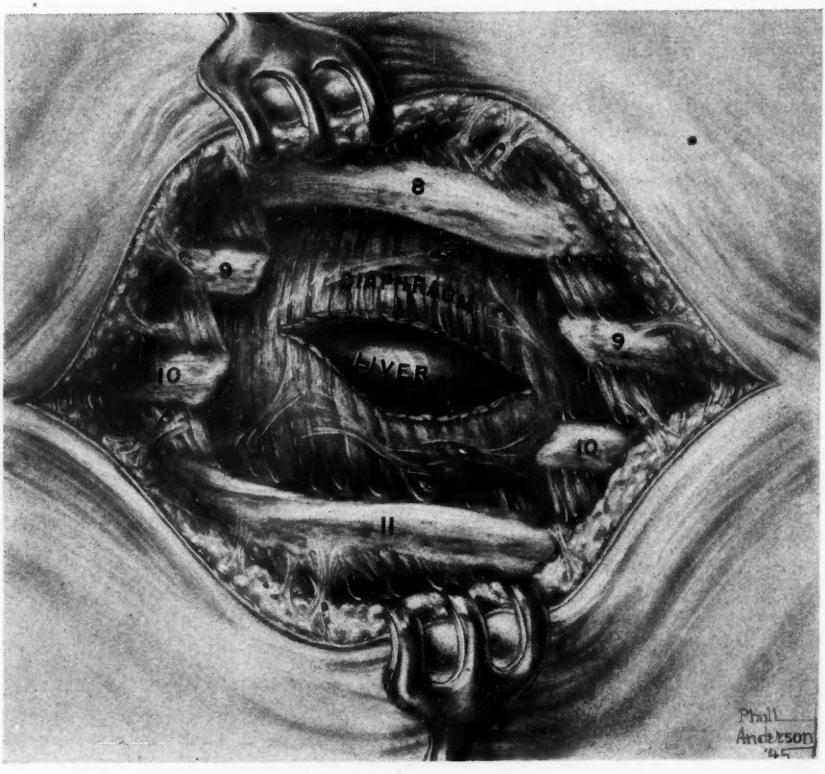


Fig. 10 (Case 10).—Drawing illustrating the findings at operation. Note liver presenting through incision in diaphragm in midportion of defect and absence of segments of ribs nine and ten.

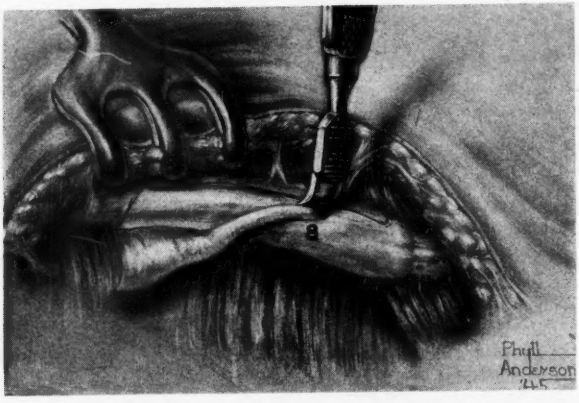


Fig. 11 (Case 10).—An illustration of method used in preparing a periosteal flap from the eighth rib.

portion of the defect traversed the diaphragm and peritoneum, exposing the liver (Fig. 10). Coughing, produced by irritation of the carina with a soft catheter passed through the endotracheal catheter, caused the liver to herniate through the defect. Further exploration

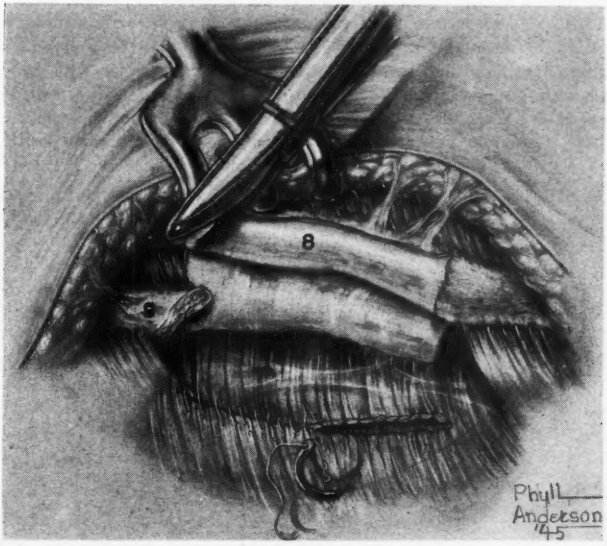


Fig. 12 (Case 10).—Drawing illustrates the method of cutting ribs tangentially to insure "locking." Note periosteal flap developed from the eighth rib. Incision in the diaphragm was closed with interrupted silk sutures.

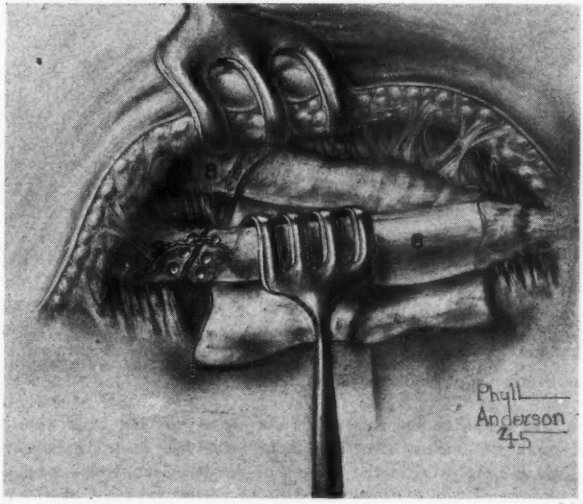


Fig. 13 (Case 10).—An illustration of the method used to pull the eighth rib into position over the defect and fix it in place with No. 1 chromic catgut sutures placed through small drill holes in corresponding positions at the end of the displaced eighth rib and the posterior stump of the ninth rib.

was discontinued and the incision in the diaphragm was closed with interrupted medium silk sutures. In order to bridge the defect satisfactorily, the eighth rib was cleaned of periosteum (Fig. 11) and cut in the posterior axillary line (Fig. 12). Drill holes were made for three No. 1 chromic catgut sutures in the posterior extremity of the cut portion of the eighth rib. These sutures were then brought through corresponding drill holes in the anterior stump of the ninth rib and tied in place (Fig. 13). The eleventh rib was then cut in the anterior axillary line and prepared in the same manner with three small drill holes threaded with No. 1 chromic catgut sutures which were then threaded through the anterior stump of the tenth rib. The two rib ends were cut on a bias in such a manner as to prevent the "springing back" of these structures into their normal position (Fig. 14).

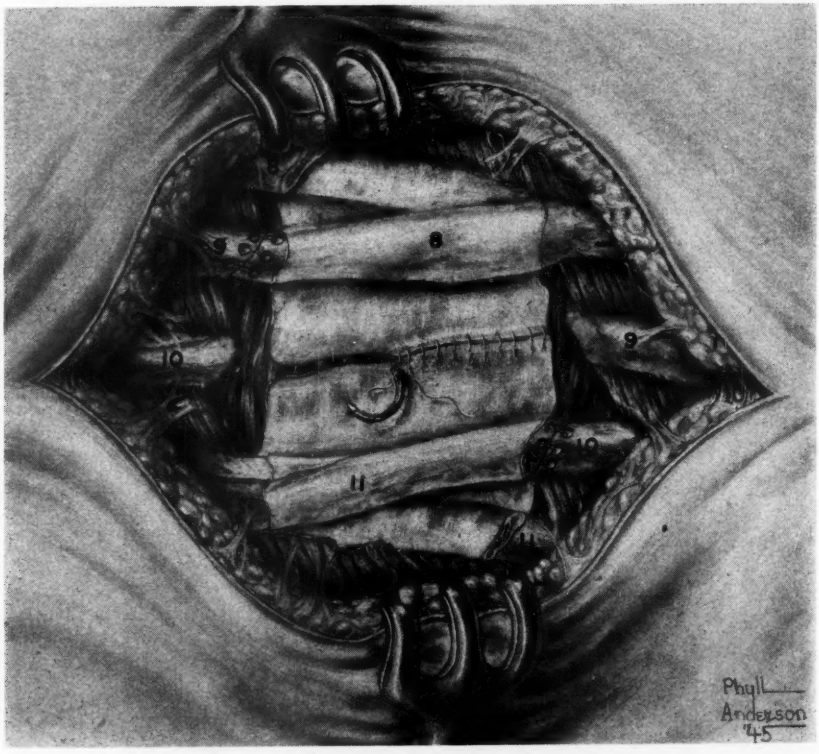


Fig. 14 (Case 10).—Drawing illustrating the completed operation. The eighth and eleventh ribs have been cleaned of periosteum and pulled into position over the defect. Note method of immobilizing the displaced ribs accomplished by cutting through the ribs tangentially and fixing them to the rib stumps of the ninth and tenth ribs with No. 1 chromic catgut sutures placed through drill holes. The periosteal flaps were approximated under tension over the central portion of the defect.

In the course of the operation the muscles of the chest wall and back were necessarily cut across. Small openings in these structures were made by sharp dissection and the ribs, namely, the eighth and eleventh, in the process of displacement were brought through these apertures. This produced a flat chest wall and permitted suturing of the open periosteal flaps in reverse manner over the midportion of the defect (Fig. 14). The subcutaneous tissue and skin were closed with interrupted fine silk sutures. A dry sterile compression dressing was applied.

The patient's postoperative convalescence was entirely uneventful and the wound healed per primam. During the first seven days following operation the soldier complained only of feelings of insecurity at the site of operation on deep breathing or coughing. Examination Aug. 21, 1945, three weeks following the procedure, showed the chest wall to be rigid. The patient at this time was entirely without complaints and could not produce any herniation with Valsalva's maneuver, coughing, or straining (Fig. 9, *B*).

Comment.—In Cases 8, 9, and 10, defects of the chest wall, associated with hernia of the liver, were satisfactorily treated and evidently cured by the use of structures of the chest wall in and about the margins of the defect (rib, periosteum, and muscle).

CASE 11.—A 30-year-old soldier sustained a penetrating wound of the left chest in the midaxillary line at the level of the ninth rib July 30, 1944, when a truck in which he was riding accidentally collided with another truck. According to the history, a bamboo pole was thrust forcibly through the left chest, producing fractures of the eighth and ninth left ribs in the midaxillary line. The patient was in shock, was dyspneic, and expectorated blood. At an overseas hospital, numerous thoracenteses were performed with removal of blood from the left pleural cavity. The soldier returned to the United States in November, 1944, and resumed duties as a truck driver. While working in this capacity in April, 1945, the patient first noticed a bulge in the left chest at the level of the ninth rib. The bulge continued to enlarge and was associated with pain over the left lower chest on coughing, sneezing, and deep breathing. The soldier was no longer able to continue his work as a truck driver because of the pain produced by the motion of the truck. Accordingly, he was admitted to a station hospital in New York, where a diagnosis of hernia of the thoracic wall, left, was made. He was transferred to Walter Reed General Hospital Aug. 5, 1945, where examination revealed a soft, tender bulge in the ninth interspace in the midaxillary line which appeared on performing Valsalva's test or coughing and disappeared on quiet breathing or inspiration. The bulge at the height of forced expiration, with the glottis closed, measured 6 by 5 cm. in diameter and appeared through a defect in the ninth interspace which measured 6 by 3 cm. in diameter. The remainder of the physical examination was not remarkable. As in the three previous cases, it was impossible to tell before operation the exact nature of the contents of the thoracic bulge.

Operation.—On Aug. 24, 1945, under endotracheal oxygen-ether anesthesia, an incision was made over the defect in the left chest wall. The subcutaneous tissues and muscles were divided, exposing the bony thorax. There was a defect in the ninth interspace in the midaxillary line measuring 6 by 3 cm. in diameter. The intercostal muscles were markedly attenuated. Incision through the midportion of the soft tissue bulge exposed peritoneal cavity. A portion of the omentum was adherent to the margins of the defect and was released by sharp dissection. Periosteal flaps were developed from the external surfaces of the ninth and tenth ribs and closure of the defect was effected by suturing the intercostal muscles with interrupted medium silk sutures. This was reinforced by approximating the periosteal flaps from the ninth and tenth ribs over the midportion of the defect with interrupted medium silk sutures. The muscles of the chest wall were closed in layers with interrupted fine silk sutures. The skin was closed without drainage.

Postoperative convalescence was uneventful. Examination Sept. 1, 1945, when sutures were removed from the wound, showed no evidence of bulge. Re-examination one month following operation showed the area of defect to be covered by a firm plate of tissue which had the consistency of bone.

Comment.—In this case it was impossible to determine before operation the exact nature of the contents of the thoracic bulge. However, the defect was in the midaxillary line in the ninth interspace and, in light of our previous experiences with defects near or below the ninth rib, as in Cases 8, 9, and 10, we anticipated finding a communication with the peritoneal cavity.

SUMMARY

1. Eleven cases of defects of the chest wall are presented.

2. Seven cases of hernia of the lung are reviewed. In three cases the liver was in the hernial sac; in one instance, the omentum.

3. Techniques for the repair of defects of the chest wall are described.

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INTRAPERICARDIAL APPROACH TO THE LUNG ROOT IN THE TREATMENT OF BRONCHIAL CARCINOMA BY DISSECTION PNEUMONECTOMY

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IN RELATION to sepsis, the pericardial sac has been in the position that first the peritoneum and then the pleura were in years ago. In the service of most thoracic surgeons there are probably examples of injury to the pericardium, either accidental or intentional, during a dissection pneumonectomy for bronchial carcinoma, and occasionally deaths from suppurative pericarditis have occurred. There is good reason to believe, however, that the pericardium possesses powers in dealing with infection at least as great as those of the peritoneum as long as mechanical factors such as the accumulation of pus under tension are avoided. A technique of individual ligation of pulmonary vessels after wide opening of the pericardium has therefore been evolved, with the result that those bronchial growths closely set between the hilar structures, those surrounding or even adherent to the pulmonary vessels, and those in which the primary growth or secondary glands are adherent to the pericardium can now be dealt with more radically. The operations were undertaken in the belief that the pericardium was no bogey for infection, and the few results recorded indicate that this faith is well founded.

When the growth is peripherally placed and the hilar structures are separated by soft glands and areolar tissue, the usual dissection outside the pericardium presents no difficulties. If vessels are felt stretched over masses of growth, or if neoplastic tissue is felt so near to the pericardium that a good margin of tissue cannot be obtained beyond it, intrapericardial dissection offers a quicker, safer, easier, and, at the same time, more radical operation.

ANATOMY

Only certain points in the anatomy of the pericardium need to be mentioned. The tough fibrous sac is continued over the large vessels which pass through it and merges with their outer wall. This can be clearly seen in the two branches of the pulmonary artery. The right and left pulmonary arteries are really outside the serous pericardium, but they receive contributions to their wall from the fibrous sac, and their inferior aspects bulge into the serous sac. A variable amount of their anterior and posterior aspects are also covered with serous membrane which forms a fornix where it is reflected off the vessels in front and behind. If the line of fusion of visceral and parietal pericardium on the superior vena cava is followed, it is found to pass outward and downward on the vessel. It then passes around the outside to the posterior aspect (Fig. 1). The lower end of the superior vena cava is almost entirely within

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the sac. If the finger be passed downward and outward on the serous-covered vena cava, it falls backward onto the superior pulmonary vein. There is, however, a depression in the angle between these two veins, and it is into this that the right pulmonary artery bulges (Fig. 1). This depression is of some descriptive importance and will subsequently be referred to as the postcaval recess. It is bound in front by the vena cava, below by the superior pulmonary vein, and above by the right pulmonary artery. The recess is easily opened by retracting the vena cava forward and medially and dividing the serous reflection which passes between the posterior surface of the cava on to the

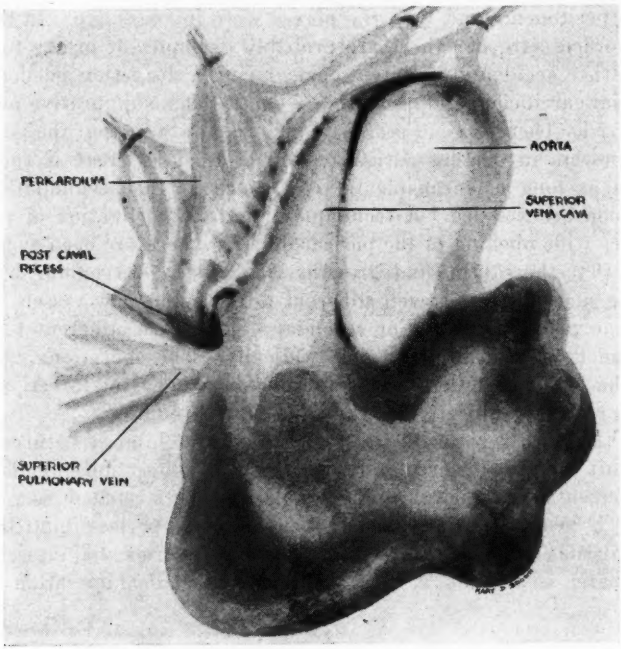


Fig. 1.—Serous pericardium opened in front and retracted to show the reflection of parietal and visceral pericardium on the superior vena cava and the postcaval recess.

anterior aspect of the right pulmonary artery (Fig. 2). If this is done, the finger may then be passed backward and downward over the top of the right pulmonary artery, the fibrous expansion from the pericardium onto this vessel being divided or displaced, and it will appear in the floor of the postcaval recess covered by a single layer of serous pericardium. Division of this layer frees the artery from its venous attachments and leaves its whole circumference and a considerable length free for ligature and division.

When the pericardium has been opened at the front and on the right side, the superior and inferior pulmonary veins are seen entering the atrium of the auricle. The veins are of variable length. They may unite immediately after entering the pericardial sac, or there may be an interval between them through

which the finger can be passed after division of the double membrane which unites them. It is not possible on the right side to pass the finger backward below the inferior pulmonary vein, as a double reflection of serous pericardium descends from the vein to envelope the inferior vena cava. If this double layer is divided, however, the finger can then be passed upward in the oblique sinus behind the right pulmonary veins. It may emerge covered by two serous layers either between the two pulmonary veins or higher up between the superior vein and the right pulmonary artery in the postcaval recess. In either case, the two layers must be divided to free the circumference of the pulmonary veins for ligature.

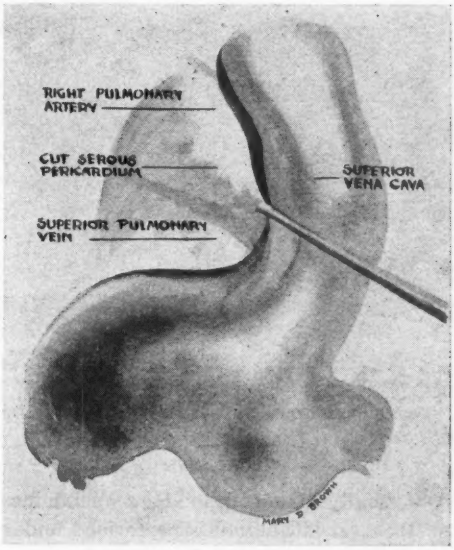


Fig. 2.—Postcaval recess opened by retraction of superior vena cava and division of serous pericardium between this and the right pulmonary artery. This shows the artery bulging into the serous pericardium with its lower aspect covered and its upper part outside the pericardium.

On the left side the left pulmonary artery bulges into the top of the serous pericardium as on the right, but here the bulge is more obvious, for it is not crossed by anything corresponding to the superior vena cava; there is, however, a small recess between the artery above and the superior pulmonary vein below. The recess is bound medially by a ridge passing from the main pulmonary artery to the superior pulmonary vein, known as the vestigial fold of Marshall. The two pulmonary veins are here more widely spaced than on the right side so that individual ligation is easier. On the left side there is no double reflection of pericardium below the inferior vein. The finger can be passed backward and upward into the oblique sinus behind the pulmonary veins without hindrance. It may then appear through the double layer be-

tween the two veins, or it may pass higher up and present between the upper vein and the left pulmonary artery. If it should be necessary, the upper vein may be still further freed by dividing the vestigial fold of Marshall. The appearances of these structures from the front are shown in Fig. 3.

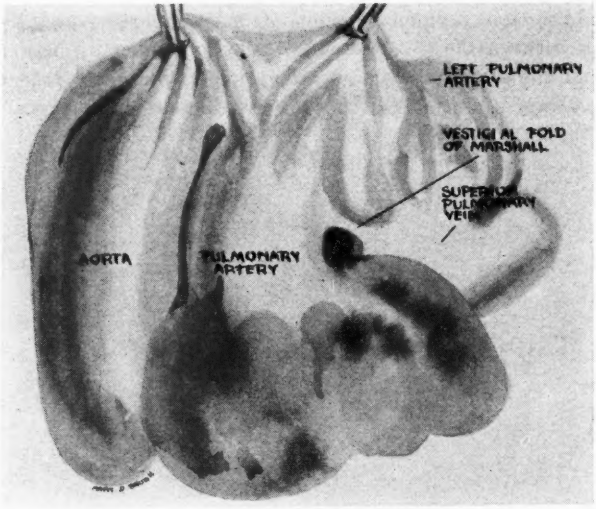


Fig. 3.—Reflections of serous pericardium on the left side showing continuous smooth layer covering the under aspect of the left pulmonary artery and sweeping on to the anterior aspect of the superior pulmonary vein. Note the vestigial fold of Marshall extending between this vein and the main pulmonary artery.

TECHNIQUE

Premedication is usually omnopon, gr. ⅓, scopolamine, gr. $\frac{1}{150}$, and atropine, gr. $\frac{1}{100}$. Intratracheal intubation is performed under a local anesthesia. All the patients are given continuous blood drip during operation. Anesthesia is maintained by sodium pentothal into the blood drip together with nitrous oxide and oxygen through the tracheal tube. Cyclopropane is also used.

The chest is opened by either of the two recognized approaches for lung carcinoma; the anterior incision with removal of as much of the second, third, or fourth ribs or cartilages as may be required, or the posterolateral incision with removal of the whole length of one or more ribs. In a general way, the posterolateral approach is preferable for this gives a good exposure of the front and back of the lung root and allows the bronchus to be isolated and occluded at the earliest possible stage in the operation. The immediate results in the surgical removal of the lung for carcinoma depend very much on the question of spillover of septic secretions into the good lung, and it is therefore a great advantage if the affected bronchus can be occluded either by clamp or strong ligature before much manipulation of the lung takes place. More often this can be done easily by the posterolateral approach.

The intercostal nerve or nerves involved in the incision are divided and the corresponding vessels ligatured and divided before the pleura is opened. After a preliminary exploration to determine as far as possible the operability of the growth, the lung root above and behind is infiltrated with local anesthetic. The dorsal lobe is carefully mobilized and the mediastinal pleura behind it divided. The posterior aspect of the bronchus is thus exposed. Here bronchial vessels may have to be divided. By a combination of sharp and blunt dissection, the circumference of the main bronchus may be freed enough for a stout colostomy silk ligature to be passed around it and tightly tied. This must be above any palpable growth, but otherwise it need have no relation to the point at which ultimate division of the bronchus is to be made. It is a purely temporary measure to prevent the spillover of secretions. Adhesions may then be divided. The lateral pulmonary ligament is severed between ligatures up to the lymphatic gland, which lies below the inferior pulmonary vein. This gland should be removed with the lung at a later stage. The division of the main pulmonary vessels may now be started.

Left Side.—The left phrenic nerve is crushed as it crosses the aortic arch. An incision about two and one-half inches long is then made in the pericardium immediately in front of, and parallel with, the phrenic nerve. This incision is extended backward over and below the lung root, dividing the phrenic nerve and accompanying vessels which have been understitched and tied. When the outer edge of the pericardium is retracted, the most obvious vessel is the anterior superior pulmonary vein. The reflection between the two veins is easily perforated with curved scissors and the opening enlarged so that the finger can be passed around the vein. The vein should then be hooked forward while the serous reflections are still further divided, both toward and up to the parietal pericardium and inward to the auricle. In this way the length of vein available for ligature and division inside the pericardium is ample, and there should be no fear of cutting through a ligature. My practice has been to apply No. 6 silk ligatures as far apart as possible, and then to transfix the vein between the ligatures with silk stitches, one stitch adjacent to each ligature. The transfixion stitches are tied, passed around the vessel, and tied again in the same way as in the treatment of a hernial sac. The vessel is then divided between the stitches. The inferior vein is similarly treated.

The pulmonary artery is isolated next. Only a part of the under aspect of the pulmonary artery on each side is covered by serous pericardium. Its isolation, however, is much simpler when the pericardium is opened. The pericardial incision is prolonged upward to the anterior aspect of the artery. The finger can then be passed around the superior and posterior aspects of the vessel to show below covered by the serous reflection. This reflection is divided and the artery is then free to be treated by double ligature and stitching as already described for the vein. On the left side the inferior vein may present some difficulty if the anterior approach is used, for it is overlaid by the ventricles, but on the right side it is easily accessible by either approach. The fibrous pericardium over the left main bronchus is then incised. The

glands and fibrous tissue from the bifurcation of the trachea, and from within the arch of the aorta, are drawn down. The bronchus is divided and closed by a single terminal continuous stitch. The remaining pericardium behind the lung root is then divided and the lung is removed. This last incision should be well toward the midddle line so that the mediastinal tissues between the pericardium and the esophagus can be stripped down in continuity with the glands at the bifurcation of the trachea.

Where a growth occurs in the left upper lobe, and especially when the posterolateral approach is used, it may be easy first to cut the lateral pulmonary ligament and then to divide the posterior inferior vein outside the pericardium. The pericardial sac is then incised as already described and the artery and remaining vein secured within it. If malignant tissue involves the superior vein, it may be necessary to dissect the vein and the upper border of the auricle off the main pulmonary artery and to make the section through the auricle itself rather than divide the veins separately. In this event it is useful to tie a ligature of stout colostomy silk around the isolated part of the auricle. Three traction sutures of fine silk are then inserted in the auricular wall, distal to the ligature. Clamps are applied still more distally and the auricle is divided between the clamps and the traction sutures and finally stitched up with continuous fine silk. The traction sutures may then be lightly tied or removed.

Right Side.—For growths of the right lung only minor differences are to be observed. The incision of the pericardium in front of the lung root passes backward above, dividing the phrenic nerve and vessels and reaching the angle between the superior vena cava and the main right pulmonary artery. Here the serous coat is reflected on to the undersurface of the artery, leaving a fornix in front and behind. The fibrous pericardium, as it splays out in the angle between the two vessels, is divided, and the serous reflection of the anterior fornix is cut. The finger can then be passed through the posterior fornix into the postcaval recess and a long length of pulmonary artery exposed. It is doubly ligatured, stitched, and divided. The pulmonary veins on the right side sometimes join so soon after entering the pericardium that they are more easily treated as a single vessel. The vessel is really a part of the left atrium and has a fairly thick muscular wall. Below the inferior pulmonary vein the double layer passing on to the inferior vena cava is divided. Access to the posterior surface of the atrium is then possible, and after the serous reflections above the superior vein have been divided, it can be treated in the manner described for the left side. It may be occluded first by a stout silk ligature and then carefully sutured by one or more fine silk stitches. It may be necessary, in order to leave enough room for division, to omit the distal ligature and apply a clamp. In such circumstances it has usually been considered wise to tie off the pulmonary artery before dividing the veins, so that if the clamp slips, the loss of blood is only that which would be removed in the lung.

In general, the order in which vessels are tied is taken as a matter of convenience. The usual method in extrapericardial dissection is to divide the

artery first. This diminishes the amount of blood removed with the lung. On the other hand, it seems reasonable to argue that blood can very easily be replaced, and that ligature of the veins first minimizes the risk of operative trauma, forcing emboli of malignant cells into the circulation.

On either side no attempt is made to close the pericardial sac. It is considered an essential part of the operation that the pericardial sac be left in wide communication with the pleural space, into which it drains freely. This applies whether or not contamination of the pleura is thought to have occurred. It may be found necessary to insert one or two sutures to prevent prolapse of the heart or part of the heart into the pleura, otherwise impairment of cardiac function might occur from the strangulating action of the cut edge of the pericardium.

The most important points in bronchial closure are now considered to be section of the bronchus as close as possible to the trachea, simple terminal suture, and a good cover of pleural membrane with mediastinal tissue which must be stitched to the stump well above the line of bronchial suture. In the earlier cases recorded, these three points have not always been fully appreciated.

CASE REPORTS

CASE 1.—E. F. D., a merchant seaman aged 40 years, was admitted to the Infirmary with a diagnosis of squamous carcinoma of lingular bronchus, left lung. It was discovered at operation, November, 1942, that the lingula was collapsed and that the rest of the upper lobe was emphysematous. There were soft glands overlying the left pulmonary artery. One hard gland 1 cm. diameter was attached to the pericardium over the superior pulmonary vein. The primary growth extended along the superior vein up to, but not through, the pericardium. The lung was removed by intrapericardial dissection, the vessels being taken in the following order: artery, superior vein, inferior vein. The pericardium was left widely open and the chest closed without drainage. Three weeks after operation he developed a bronchial fistula. The pleural space was drained and then closed by thoracoplasty.

Two and one-half years later he reported having sailed to Iceland on three occasions in his old job as skipper aboard a fishing trawler. He was perfectly fit and well, and there was no evidence of recurrence on physical or radiologic examination.

Pathologist's Report.—The specimen consisted of the whole of the left lung with an area of pericardium attached. The lingular bronchus was occluded by a very active and cellular squamous carcinoma. The lingula itself was collapsed and bronchiectatic, and the upper lobe showed obstructive emphysema. The growth was very intimately apposed to the anterior aspect of the superior pulmonary vein and formed a hard bulge between this and the inferior vein where it was adherent to the pericardium. When the pericardial aspect was shaved off, this bulge between the two veins was seen to be a mass of growth which had extended into the lower lobe. The bronchus had been divided well above the growth, but the hilar lymph glands attached to the specimen showed involvement by neoplastic tissue.

CASE 2.—A. E. F., a man aged 38 years, was admitted to the Infirmary with a diagnosis of highly malignant oat-cell carcinoma, right lower and middle lobe bronchi. At operation, May, 1943, dense adhesions were found over the lower and middle lobes. The right main pulmonary artery was short. Its two branches were divided outside the pericardium, before a hard nodule was felt on the posterior aspect of the superior pulmonary vein. The dissection was therefore completed intrapericardially. In order to avoid malig-

nant tissue, the auricle itself had to be divided. On the tenth day a bronchial fistula developed, and the pleural space was drained. On the twentieth day he developed symptoms of meningitis and died in twenty-four hours.

Post-mortem examination showed an extensive purulent meningitis, infection of the right pleural space, and a wide open bronchus. The cut edge of the pericardium was adherent to the heart all around, and the pericardial sac was obliterated by fibrinous adhesions. No infection was present. The auricle was firmly healed, its inner wall being perfectly smooth and free from thrombus.

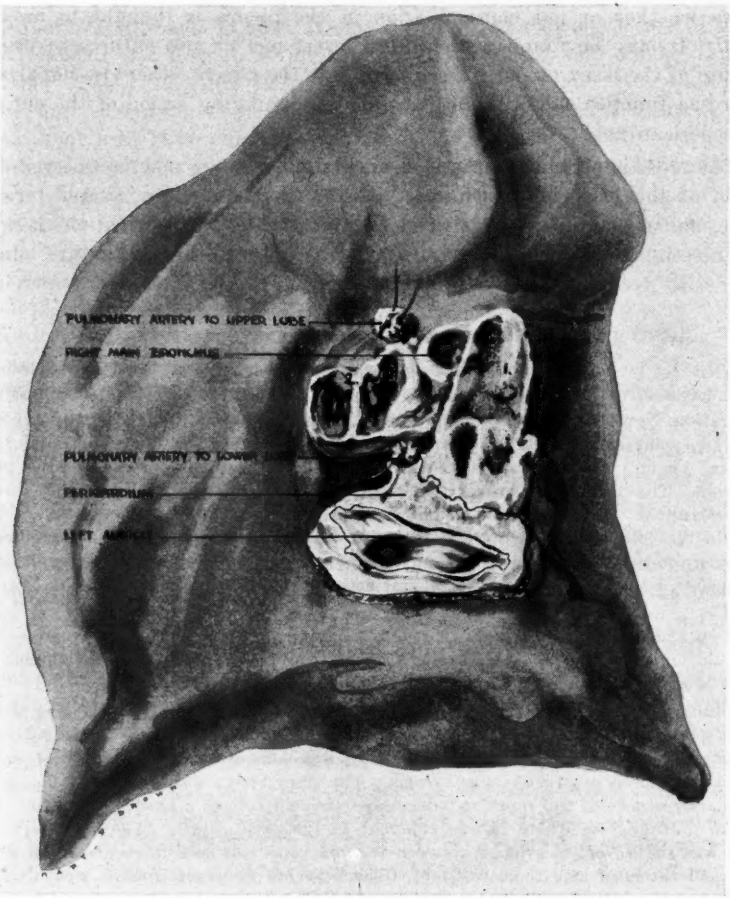


Fig. 4 (Case 2).—Carcinoma of right lower and middle lobe bronchi occluding inferior pulmonary vein and bulging into the pericardium between the two veins. Secondary deposits in lymphatic glands *1* and *2*.

Pathologist's Report.—The specimen consisted of the whole right lung with part of the left auricle and pericardium attached. The section of auricle measured 3 cm. in length and 1 cm. in width and was in continuity with the two right pulmonary veins (Fig. 4). Immediately above it was a group of three lymphatic glands extending up to the bifurcation of the trachea. These were heavily anthracotic but also contained unmistakable new

growth. The glands in front of the bronchus around the pulmonary artery were also involved. Section of the lung showed a mass of cancer in the hilum of the lower lobe, closely investing the pulmonary veins, occluding the bronchi of the lower and middle lobes, and extending up the bronchus as a pedunculated mass. The lower and middle lobes showed advanced bronchiectasis.

Histologic examination showed that, in the main, the tumor was an oat-cell carcinoma of great cellularity, containing numerous mitotic figures. In places, however, it was distinctly of polymorphous cell type, with small giant cells, and suggestive of an anaplastic carcinoma.

CASE 3.—J. P., man aged 50 years, was admitted with a diagnosis of squamous carcinoma, right dorsal lobe. Treatment was begun in November, 1943. In view of a low blood count and very toxic appearance, three pints of blood were given Nov. 6, 1943. Nov. 8, 1943, he had a rigor. As no cause for this was apparent, the operation was carried out as planned Nov. 9, 1943. There was a massive growth in the dorsal lobe, adherent to parietal pleura and posterior pericardium. The parietal pleura over the dorsal lobe was dissected off the chest wall and removed with the lung. The root vessels were divided intrapericardially in the following order: right pulmonary artery, auricle. The pericardium was stitched from the level of the inferior vena cava up to the level of the lower end of the auricular stitch. The chest was drained for forty-eight hours. The following day the temperature rose to 102° F. and it became obvious that the pleura and chest wall were infected, and drainage had to be established again. Temperature continued to fluctuate and twelve days after operation the patient had another rigor. Blood culture showed the presence of *Staphylococcus aureus*. Rigors were repeated until death occurred Nov. 29, 1943, twenty days after operation.

Post-mortem examination showed pyemic abscesses in both kidneys and a patchy purulent meningitis. The cut edge of the pericardium was everywhere adherent to the heart so that the pericardial cavity was completely sealed off from the pleura. The pericardium contained a trace of clear yellow fluid, but no signs of infection.

Pathologist's Report.—The specimen consisted of the whole right lung with a portion of pericardium attached surrounding a part of the left auricle. The dorsal lobe was occupied by a necrotic neoplasm 7.5 by 7.3 cm. in diameter, and over this a flap of parietal pleura was adherent.

Histologic section showed it to be a squamous carcinoma with extensive keratinization. Widespread bronchiectasis was present in the basal lobe. The lymph glands from the bifurcation of the trachea and from in front of the lower end of the trachea showed very active hyperplasia but no neoplastic invasion.

CASE 4.—J. S., man aged 43 years, was admitted with a diagnosis of squamous carcinoma, left upper lobe. At operation, November, 1943, a large mass was found extending across the interlobar septum among the pulmonary vessels so that the superior vein was tightly arched over it. Separation outside the pericardium was impossible. The pericardium was therefore opened in front and the vessels treated as already described in the following order: superior vein, artery, inferior vein. The pericardium was left widely open, and the chest was closed. A bronchial fistula developed one week after operation. The pleura was drained. Ten weeks after operation a thoracoplasty was done to close the pleural space. The patient remained well and carried on his work as a taxi driver for ten months. He then developed dysphagia and died a little over twelve months after operation, with a mass of malignant tissue obstructing the esophagus. At post-mortem examination the pericardial sac was found to be completely obliterated.

Pathologist's Report.—The specimen consisted of the whole left lung with an area of pericardium attached around the pulmonary veins. There was a tumor in the hilum measuring 7.5 by 7 cm. in diameter. This was mainly in the upper lobe but partly extended between the vessels into the lower lobe across the septum. The upper lobe was collapsed

and the lingula in particular was grossly bronchiectatic. The tumor was a squamous carcinoma. Glands examined from below the inferior pulmonary vein, from above the left main bronchus behind the pulmonary artery, and from the bifurcation of the trachea showed no evidence of secondary deposits.

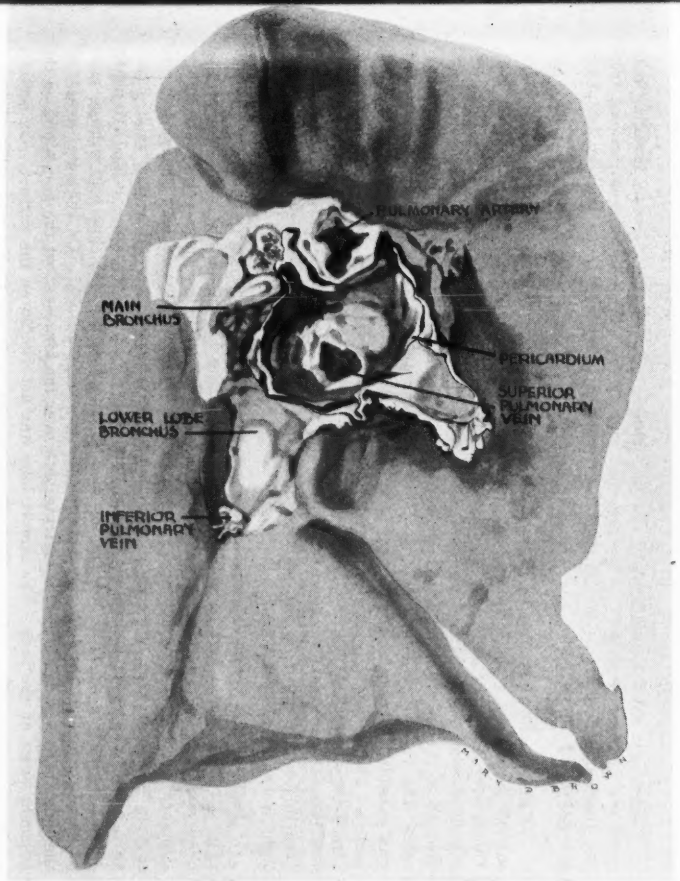
CASE 5.—G. F., a regimental sergeant-major aged 43 years, was admitted with a diagnosis of oat-cell carcinoma, right middle lobe. At operation, May, 1944, the mass in the middle lobe was found to be adherent to the pericardium. Lying on the pericardium, immediately behind the phrenic nerve and in front of the pulmonary artery, was a gland which seemed to be involved by growth. The pericardium was opened and the right pulmonary artery divided. The veins were too closely applied to the growth to be taken separately and the left auricle was therefore divided. The pericardium was left widely open and the chest closed. The patient made a normal recovery. No fistula developed, but the pleural space became infected and was treated by penicillin instillation. He was discharged home July 19, 1944. Sept. 30, 1944, he was admitted to another hospital with acute right-sided heart failure and died. Post-mortem examination showed the pericardium to be everywhere adherent and to be invaded by malignant tissue over the region of the right ventricle. The upper part of the pleural space was occupied by a mass of carcinomatous tissue in which was a cavity containing 2 ounces of pus.

Pathologist's Report.—The specimen consisted of the whole right lung with a large area of pericardium attached to it. The pericardium was stretched over a mass of growth which occupied the middle lobe. Part of the left auricle had been resected. There was obvious involvement of the glands around the main bronchus. On section, the growth projected into the basal bronchus as a polyp. There was marked bronchiectasis of the lower and middle lobes. The histology of the tumor was of a typical oat-cell carcinoma with widespread necrosis. One mass of growth in a bronchiectatic cavity had more the appearance of a spheroidal cell carcinoma. Invasion of hilum glands was confirmed.

CASE 6.—W. L., a sailor aged 36 years, was admitted to the Infirmary with a diagnosis of oat-cell carcinoma, left upper lobe. At operation, August, 1944, the growth was found to be very extensive within the arch of the aorta. It was adherent to the pericardium in the region of the anterior pulmonary vein and was bulging through the mediastinal pleura behind just in front of the posterior part of the aortic arch. Two enlarged glands were felt in the superior mediastinum above the aorta. The growth was first carefully separated from the aortic arch. The mediastinal pleura was reflected down from the apex of the chest with the two superior mediastinal lymph glands attached, the phrenic and recurrent laryngeal nerves being divided. The inferior pulmonary vein was divided outside the pericardium. The pericardium was then opened and the superior vein and the artery divided in that order. The bronchus was sutured and then covered by a flap of pericardium which was reflected backward and stitched to the posterior aspect of the bronchial stump. This, of necessity, covered also the stump of the left pulmonary artery. The pericardium was left widely open and the chest closed. Recovery was uneventful. The patient was walking about the ward after two weeks. Just after four weeks he was transferred to the Deep X-ray Department. There he suddenly had a profuse hemoptysis and died in less than a minute.

Post-mortem examination showed the pericardial sac completely closed off and clean. The pleural space was clean. The flap of pericardium was adherent to the posterior aspect of the bronchial stump, but it had approximated the stump of the left pulmonary artery to the end of the bronchus, and a fistula had developed between the two. A gland was found, left behind in the superior mediastinum, and it was invaded by growth.

Pathologist's Report.—The specimen consisted of the whole left lung with many lymph glands and an area of pericardium attached (see Figs. 5 and 6). The hilum was occupied by a mass about 5 by 4 cm. on section. The bronchus showed early dilatation. Lymph



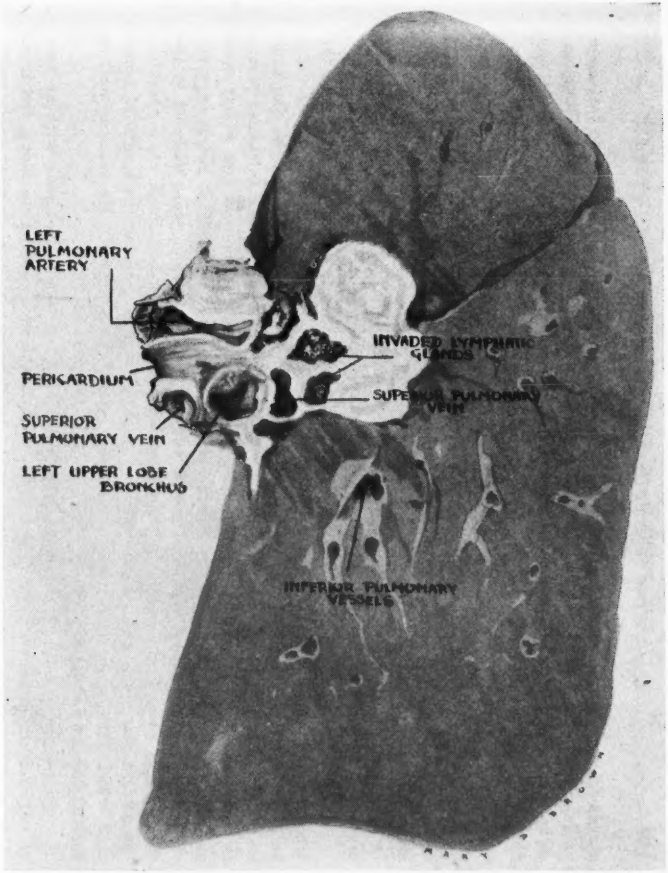


Fig. 5 (Case 6).—Carcinoma of left upper lobe bronchus compressing the right pulmonary artery and bulging into the pericardium in the region of the superior pulmonary vein. The inferior pulmonary vein has been tied outside the pericardium.

Fig. 6 (Case 6).—Section through lung to show growth in upper lobe bronchus adjacent to superior pulmonary vein, with extension round the pulmonary artery.

glands from the superior mediastinum, from the angle between the trachea and left main bronchus, and from the bifurcation of the trachea showed malignant invasion. Histologically the tumor was a typical oat-cell carcinoma, with necrosis in the primary mass, and also in the affected glands.

CASE 7.—K. B., a man aged 32 years, was admitted with a diagnosis of oat-cell carcinoma, right basal bronchus. Treatment was begun in December, 1944. About 6 ounces of turbid fluid were present in the pleura, and the lower lobe was densely adherent to the chest wall and diaphragm. The growth was very closely approximated to the pericardium in the region of the inferior pulmonary vein. The pericardium was opened and the vessels divided in the following order: artery, superior vein, inferior vein.

The posterior pericardium was incised downward and dense calcified glands from the tracheal bifurcation taken along with it. The adhesions over the lower lobe were divided and the lung removed. Adhesions were very dense over the diaphragm, and the diaphragmatic pleura had to be removed in places. Histological examination of a thickened plaque in this area showed invasion by malignant cells. The pericardium was left widely open and the chest closed. In view of the turbid fluid found in the pleura at operation, penicillin was instilled into the pleural space, and staphylococci which were cultured in the early stages soon disappeared. Recovery was uneventful and one month after operation the patient was given a full course of x-ray therapy in order to delay the spread of tumor cells which inevitably will reach the diaphragmatic lymph vessels. He remains well so far and has not developed a fistula.

Pathologist's Report.—The specimen consisted of the whole right lung with an area of pericardium attached. Old calcified tuberculous glands extended up along the inner side of the bronchus. The pericardium in the lower part was firmly applied to an underlying tumor measuring 6 by 5 cm. on section. The tumor obstructed the basal bronchus. This area of lung was densely fibrosed and bronchiectatic.

Histologic examination showed the tumor to be an oat-cell carcinoma invading both veins and lymphatics as well as the adjacent part of the lung. The plaque removed from the diaphragm was a flattened strip of dense fibrous tissue showing a striking lymphatic permeation by tumor cells.

CASE 8.—J. A. B., a schoolmaster aged 52 years, was admitted with a diagnosis of oat-cell carcinoma, right lower and middle lobes. Treatment was begun in March, 1945. There were about 10 ounces of blood-stained fluid in the pleura, and some of the basal adhesions were covered with fibrinopurulent exudate. The pericardium was opened, and through it the growth could be felt projecting into the region of the middle pulmonary vein. The veins united immediately inside the pericardium, and so no individual ligation was possible. The pulmonary artery was divided first, and then the atrium of the left auricle. The posterior pericardium was divided upward toward the carina, and a mass of glands between it and the esophagus were retracted outward and removed with the lung. The pericardium was left widely open and the chest closed. The patient made an uneventful recovery, was up and walking about after two weeks, and was discharged home eight weeks after operation.

Pathologist's Report.—The specimen consisted of the whole right lung. In the root (Fig. 7), there was a part of the left auricle measuring 1½ by ½ inch, from which three veins were seen to pass into the lower, middle, and upper lobes. These veins were tightly stretched over a mass of malignant tissue in the hyparterial bronchus. The auricle was surrounded by an area of pericardium. Immediately behind the pericardium in its lower part, and at the upper end of the lateral pulmonary ligament, was an enlarged lymphatic gland. Extending up from this on the medial aspect of the bronchus was a large mass of glands, which in the body lay between the two main bronchi. On the outer aspect of the lung there was dense pleural scarring both over the lower end of the fissure and on the adjacent parts of the lung. The middle lobe was not visible on the surface. On section,

the middle lobe was found to be completely collapsed and covered by the lower and upper lobes. Its main bronchi were packed with growth. The growth had surrounded the hyparterial bronchus and had penetrated the pericardium just above the inferior pulmonary vein (Fig. 8). Immediately above this the growth extended into the mass of glands on the medial aspect of the bronchus. Both upper and lower lobes showed bronchopneumonic changes. Histologically there was an oat-cell carcinoma invading the middle and inferior pulmonary veins, the pericardium, and the lymphatic glands at the bifurcation of the trachea.

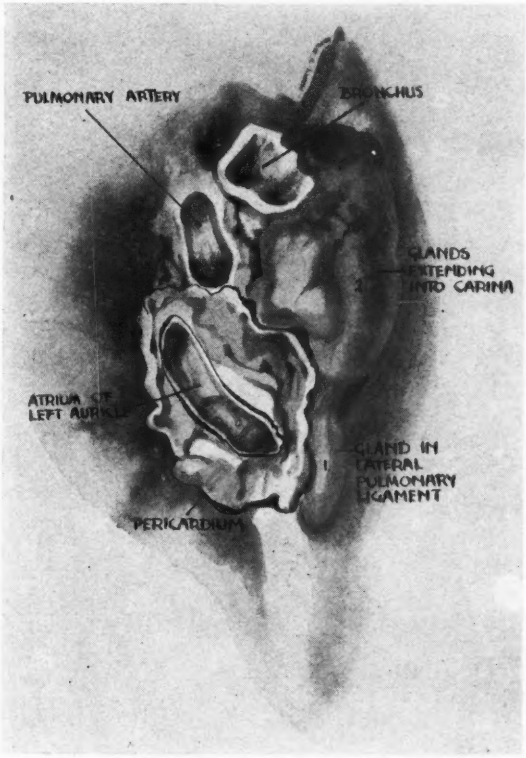


Fig. 7 (Case 8).—Hilum of right lung. Carcinoma of right lower and middle lobe bronchi, bulging into pericardium between the two main pulmonary veins. Secondary deposits in glands *1* and *2*.

Case 9.—J. S., a man aged 51 years, was admitted with a diagnosis of highly anaplastic squamous carcinoma, right upper lobe. Treatment was begun in February, 1945. The upper lobe in this patient was supplied by two separate bronchi, one in the usual situation and one coming off the right side of the trachea one and one-half inches higher. The growth occurred in the former. The lung was very emphysematous with large apical bullae. A dense, hard mass was felt in the lower part of the upper lobe. The lesser fissure was obliterated. Because of the abnormal bronchial arrangement and the presence of malignant tissue between the two upper lobe bronchi, it was not possible to tie off the bronchus first. The pericardium was opened and the vessels taken in the following order:

Fig. 8 (Case 8).—Section through lung to show the growth around the inferior pulmonary vein extending into the pericardium immediately above. Direct extension of growth into gland 2 at the bifurcation of the trachea.

inferior vein, superior vein, artery. The posterior pericardium was then incised upward, and at this stage an abnormal branch of the pulmonary artery was found. It passed from the top of the right main artery at its junction with the parent stem up to that part of the lung corresponding to the abnormal bronchus. It was divided. An incision was then made obliquely across the lower end of the trachea, from just above the abnormal bronchus to the carina, and the lung was removed. The trachea was sutured and covered with pleura. The pericardium was left open and the chest closed. The patient made an uninterrupted recovery, was out of bed in seven days, walking in fourteen, and down to the gymnasium in twenty days. He left the hospital six weeks after operation.

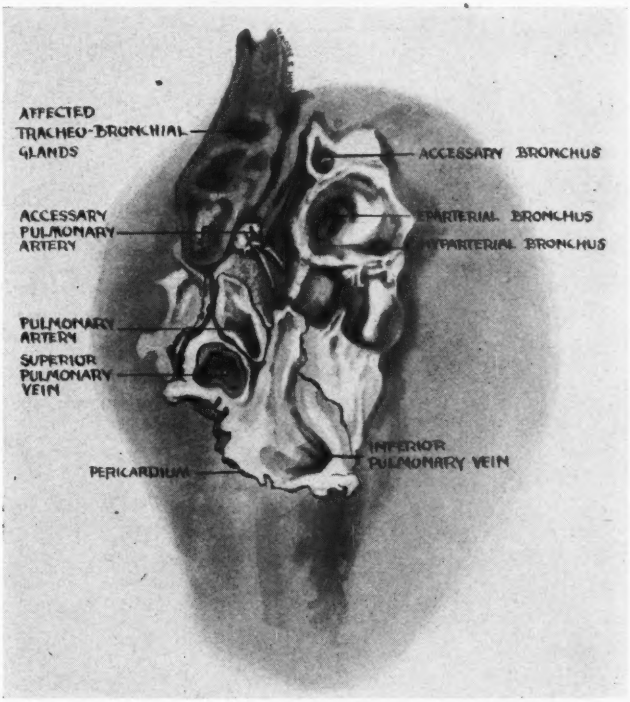


Fig. 9 (Case 9).—Hilum of right lung. Eparterial bronchus occluded by growth. In this case the intrapericardial dissection was necessary in order to tie off the pulmonary artery far enough away from the main growth and the big mass of glands immediately in front of it.

Pathologist's Report.—The specimen consisted of the whole right lung. About one-half inch above the right main bronchus was an accessory bronchus. The normal upper lobe bronchus was occupied by a growth which extended on to the lateral wall of the main bronchus. The growth occupied the space between the main bronchus and the accessory bronchus and extended into the substance of the lung for about two inches (Figs. 9 and 10). Immediately in front of the main bronchus was a mass of glands obviously invaded by malignant tissue. Closely applied to the posterior aspect of these glands was an accessory pulmonary artery corresponding to the accessory bronchus. Immediately below the glands was the main right pulmonary artery. Below this was an area of pericardium surrounding the two pulmonary veins. At the apex of the lung were numerous large emphysematous bullae, some

. . .

of which were 2 to 2½ inches in diameter. The rest of the apex, which was supplied by the accessory bronchus, showed gross emphysema, but the pectoral and subapical zones, supplied by the ordinary eparterial bronchus, were completely collapsed and showed one or two small abscess cavities. The lower and middle lobes were normal. Histologically the tumor was a highly anaplastic squamous carcinoma.

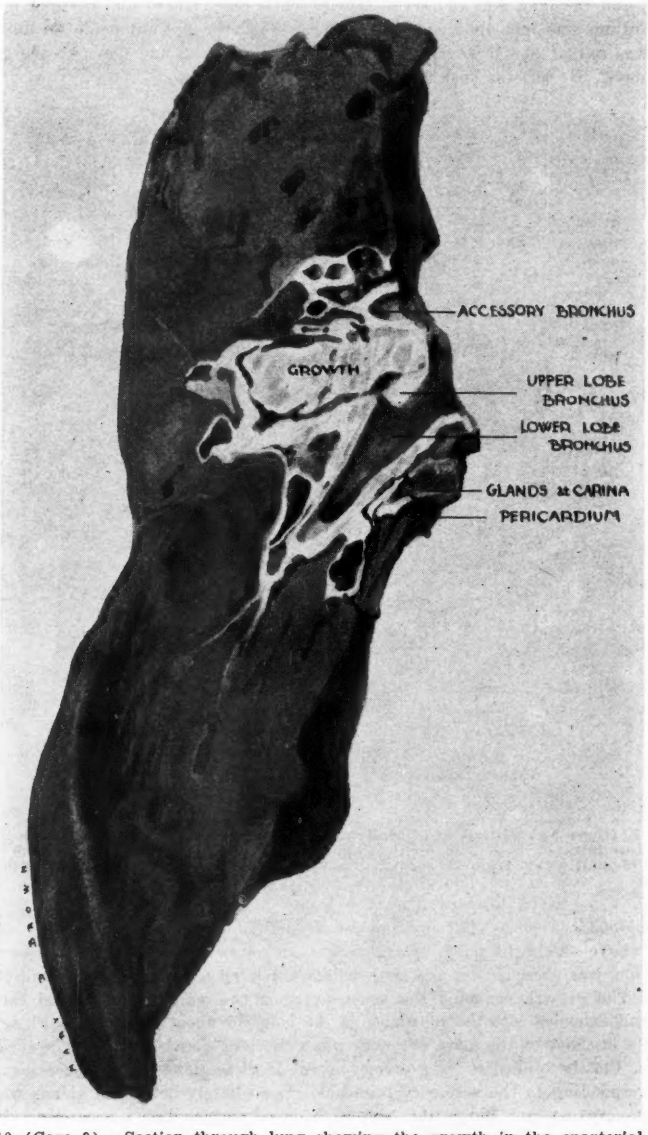


Fig. 10 (Case 9).—Section through lung showing the growth in the eparterial bronchus extending almost to the point of section. Note the accessory bronchus coming off the side of the trachea.

CASE 10.—A. G., a farm laborer aged 42 years, was admitted with a diagnosis of keratinizing squamous carcinoma, left lower lobe. Treatment was begun in March, 1945. The lower lobe was solid and turgid; the upper lobe, normal. The hilum was surrounded by glands, but many of these were felt to be soft and probably inflammatory. The pericardium was opened and the vessels taken in the following order: inferior vein, superior vein, artery. The pericardium was left widely open and the chest closed. The patient made an uneventful recovery, was out of bed on the tenth day, and was discharged from the hospital twenty-five days after operation. He remains well.

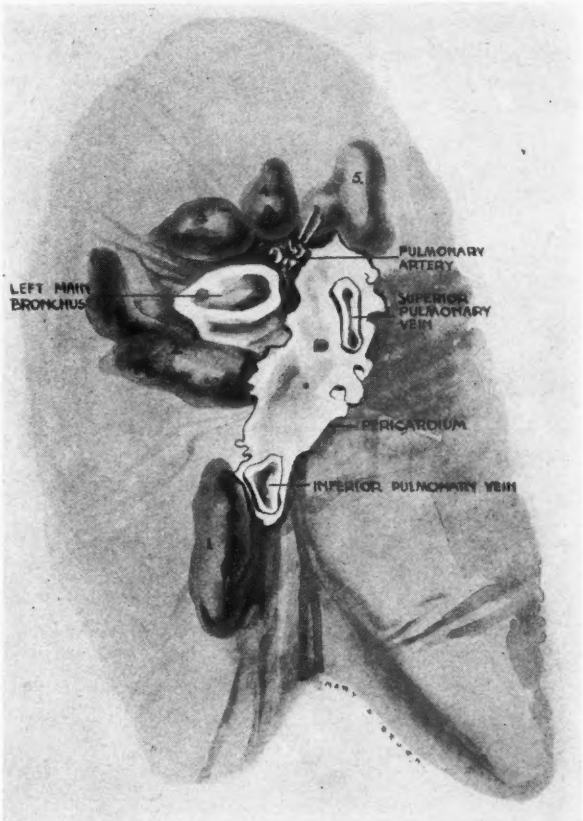


Fig. 11 (Case 10).—Hilum of left lung, showing distribution of affected lymphatic glands. The inferior pulmonary vein is almost completely occluded by growth.

Pathologist's Report.—The specimen consisted of the whole of the left lung with a mass of glands attached. At the root of the lung was a large area of pericardium extending from the pulmonary artery above to below the inferior pulmonary vein (Fig. 11). The inferior pulmonary vein was extensively infiltrated by growth and almost completely occluded (Fig. 12). The lymphatic gland below this vein was extensively replaced by growth as were also those above and below the main bronchus. The lower lobe was completely collapsed. In the dorsal lobe was a chain of abscess cavities containing pus. The

upper lobe appeared normal. Histologically the tumor was a keratinizing squamous carcinoma of the left lower lobe bronchus with extensive involvement of hilar lymphatic glands.

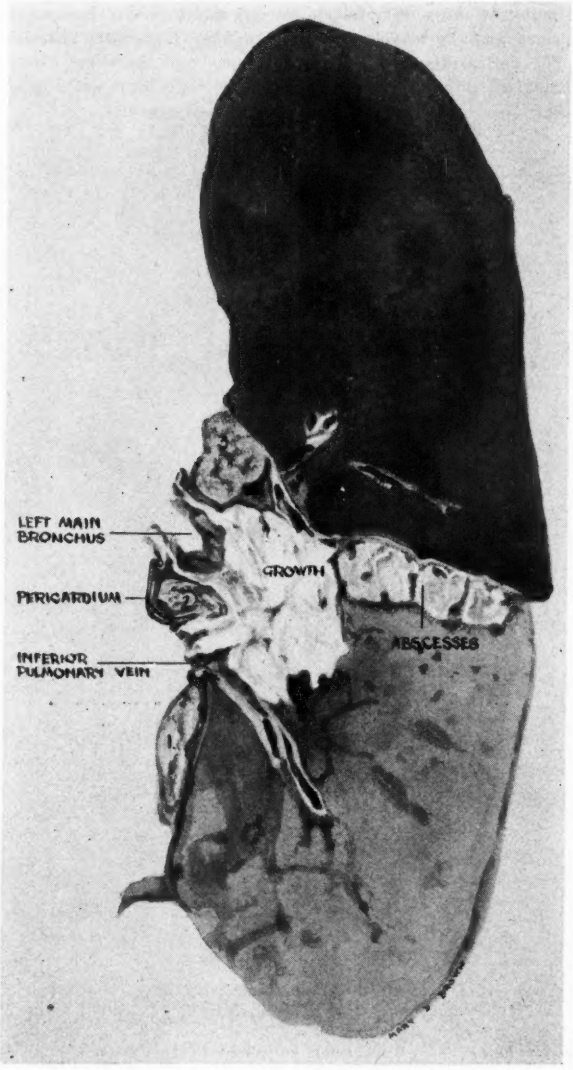


Fig. 12 (Case 10).—Section through lung to show the growth invading the pericardium and compressing the inferior pulmonary vein. Note also gland 2 just below the bronchus, projecting into the pericardium.

DISCUSSION

Any technique which increases the margin between growth and operative section in the treatment of malignant disease is worth pursuing. The intra-

pericardial dissection of the lung vessels does allow a more radical operation to be performed. It also makes for ease and speed, and in those cases where it is needed, it is a safer operation. The present recorded series of operations have been performed on patients who were otherwise considered to be inoperable, and no conclusions can be drawn about the relative merits of extra- and intrapericardial dissection in respect of end results. Sepsis seems to be no contraindication to opening the pericardium as long as it is left in free communication with the pleural space afterward. The pericardial sac appears to drain into the pleura and to become sealed off in from two to three days. Two patients in this series have died of meningitis, but in one patient (Case 3) there is reason to believe that the systemic infection occurred before operation, and in neither patient was there any sign of septic thrombus on the wound of the auricle. In one patient who has survived two and one-half years there has been no demonstrable cardiac incapacity. He has done the strenuous work of a skipper on an Icelandic trawler without any disablement.

SUMMARY

1. The anatomy of the pericardial reflections on the great vessels is briefly reviewed.

2. A technique for division of the pulmonary vessels within the pericardium is described, which is applicable to cases of bronchial carcinoma.

3. A series of ten cases of bronchial carcinoma treated by pneumonectomy with intrapericardial dissection of the pulmonary vessels is described.

TREATMENT OF PULMONARY ACTINOMYCOSIS WITH A REPORT OF SEVEN ARRESTED CASES

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PULMONARY actinomycosis has been considered a somewhat rare and almost hopeless disease, which is primarily interesting as a diagnostic curiosity. The majority of the reports on the disease are of this nature. In 1911 Harbitz and Grondahl[1] reported on 87 patients with actinomycosis in Norway in 23 per cent of whom the thorax was involved, with a 100 per cent mortality in those with thoracic involvement. Good[2] in 1930 reported on 13 patients with pulmonary actinomycosis at the Mayo Clinic, of whom 47 per cent had already died at the time of his report. He commented that "the disease is almost always essentially fatal."

Wangensteen,[3] in an excellent article in 1932, summarized all of the successfully treated cases of actinomycosis of the chest, finding 18 cured cases from 1892 to 1932, and adding one of his own. A characteristic feature is that all of the cured patients except one had some form of surgery, with extensive rib resections in 9 of them. One of these patients had remained cured for sixteen years, and another one for over six years.

With the advent of chemotherapy and penicillin during the past few years, somewhat more optimistic reports are appearing in regard to the outlook for patients with actinomycosis. Patients with cervical actinomycosis have always proved more amenable to treatment than have those with thoracic or abdominal involvement. Of the 5 patients reported by Myers[4] in 1937, 2 had involvement of the neck and chest, which improved markedly on thymol treatment following the excision of their sinus tracts. Joyce[5] in 1938 reported on 5 patients with cervical involvement who were cured with thymol following surgery. MacCharles and Kippen[6] in 1939 reported on 3 patients with face and neck involvement successfully treated with sulfonamides following surgical drainage. Bisgard[7] in 1939 reported on 2 patients in whom successful results were obtained over periods of sixteen and twenty-six months, respectively. Both patients showed the extension of the disease through the chest wall into the pleural cavity, requiring Schede thoracoplasties and repeated débridement procedures followed by irradiation and iodide administration. Miller and Fell[8] in 1939 mentioned the successful treatment with sulfonamides of a patient with extensive involvement of the bladder and abdominal wall. Wilkinson[9] reported a case in 1941 in which a cure was obtained in an 11-year-old girl with widespread involvement of the lung, chest wall, cheek, hip and foot. Surgical drainage was used in this patient, followed by an extensive course of sulfonamides over eighty-three days.

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In July, 1945, Dobson and Cutting[17] reported on sixteen patients with actinomycosis, with involvement of the lungs in 3, of the abdomen in 2 and of the cervical region in 11. Extensive treatment with sulfonamides and penicillin, in addition to surgery, produced very favorable results with seven cures, seven arrested cases and two deaths. Two of the patients with pulmonary involvement were cured by surgical drainage and penicillin. The other patient with pulmonary involvement died following a course of sulfonamide therapy without surgery or penicillin.

TABLE I. TREATMENT OF ACTINOMYCOSIS

CASE	AGE	PATIENT	APPARENT CURE	DATE OF ONSET OF DISEASE	DURATION OF LIFE AFTER ONSET OF DISEASE	CAUSE OF DEATH	EXTENT OF DISEASE	POSITIVE LABORATORY FINDINGS
1	51	A. F.	Yes	1931	9 yr.	Heart disease	Right lung and chest wall	Section of pus
2	16	B. R.		1925	2½ yr.	Actinomycosis Brain involvement	Lung, chest wall, vertebrae	Drainage smear
3	53	H. C.		1926	3 yr.	Actinomycosis	Lung, mediastinum, pleura	Post-mortem pathology
4	30	A. F.		1931	?	?	Jaw, lung, pleura	Drainage smear
5	65	C. D.		1934	3 yr.	?	Abdomen, chest, vertebrae	Smear and culture
6	34	H. E.		1936	½ yr.	Actinomycosis Brain involvement	Abdomen, chest, liver, brain	Pus culture
7	48	F. W.		1938	1 yr.	Actinomycosis Brain involvement	Abdomen, chest, brain, liver	Biopsy pathology
8	47	O. W.		1938	1 yr.	Actinomycosis	Lung, chest wall	Sputum culture
9	29	C. R.	Yes	1938	7 yr.	Living	Lung, chest wall	Sputum culture
10	29	E. D.	Yes	1939	6 yr.	Living	Lung, chest wall	Sputum examination
11	50	R. M.	Yes	1941	4 yr.	Living	Lung, chest wall	Surgical specimen drainage culture
12	18	A. M.	Yes	1942	3 yr.	Living	Lung, chest wall	Drainage culture
13	46	N. A.	Yes	1938	7 yr.	Living	Lung, chest wall, liver	Drainage culture
14	32	H. Z.		1935	8 yr.	Actinomycosis Nephrosis	Lung, chest wall	Sputum culture
15	13	J. C.		1933	½ yr.	Actinomycosis	Abdomen, thorax, liver	Post-mortem pathology
16	9	G. R.		1943	2 yr.	Living; not cured	Lung, chest wall	Sinus biopsy
17	63	J. S.		1943	6 mo.	?	Lung, chest wall	Sinus biopsy
18	21	C. G.		1938	2 yr.	Brain involvement Actinomycosis	Abdomen, thorax, brain	Sputum culture
19	17	J. L.	Yes	1939	6 yr.	Living	Lung, chest wall	Drainage culture

Table I represents a total of 19 patients with proved actinomycosis of the lung and chest wall registered at Barnes Hospital since 1925. The pulmonary involvement represented only a portion of a generalized spread of the disease in some patients. Only those in whom the diagnosis was proved by laboratory methods were included in this report. Judging by the fact that from two to three years were required to establish the diagnosis in some of the patients, who remained under treatment for that length of time, it is easy to see how a number

of positive diagnoses might have been missed. The long period of incubation required before a positive culture is identified also permits a patient to be discharged and the culture report to be omitted from the final hospital diagnosis. For these reasons, I do not believe that this represents a complete report of all the cases of pulmonary actinomycosis recorded at Barnes Hospital during the past twenty years. Apparent cures have been obtained in 7 of the patients, or 37 per cent, ranging from one year in 3 patients up to nine years in 1. No adequate record of the results of treatment is available on 4 patients, or 22 per cent. The other 8 patients, or 41 per cent, are known to have died as a result of the actinomycotic infection and its complications. One died of nephrosis, apparently as a result of the long-standing infection. Six of the other 7 patients who died had generalized actinomycosis, with involvement of the brain in 4 and of the liver and mediastinum in the other 2. The disease made its first appearance in the intestines in 5 patients, or 26 per cent, in none of whom a satisfactory result was obtained. The average duration of life following the onset of symptoms was 1.6 years in the 7 patients who died as a direct result of the actinomycotic infection.

The distribution of these patients has no apparent significance. Ten came from the state of Missouri and 6 from immediately adjacent states. Ten patients were men and 9 were women. Only 6 professed any business association with cattle, and 8 were listed as students.

A detailed report follows of the 7 patients in whom the pulmonary actinomycosis has been arrested or cured. It will be noted that surgical drainage or resection of diseased tissue was performed in all patients, supplemented by penicillin or sulfonamides or small doses of x-ray irradiation in certain instances. No potassium iodide or thymol was used in any instance. The evidence is not convincing that the x-ray therapy or sulfonamides were determining factors in the course of any of the cases reported here, although some of the patients presented subjective evidence of temporary alleviation of symptoms following couses of sulfonamides before drainage of the abscesses. It does seem, however, that penicillin did produce a remarkable effect on each of the 3 patients on whom it was used. A prompt closure of the draining chest wall sinus tracts occurred in one patient (R. M.) following penicillin administration. A reduction of marked hyperpyrexia was noted on several occasions in another patient (A. M.) following penicillin therapy. The life of the third patient (N. A.) was apparently saved on two occasions by large amounts of penicillin. The patient was practically moribund in these instances, with jaundice and dehydration following prolonged periods of pernicious vomiting and hyperpyrexia. This was accompanied by marked liver enlargement, strongly suspected of representing a liver abscess and proved to be associated with a small subdiaphragmatic abscess.

C. R., a 29-year-old man, was admitted on the Chest Service of Barnes Hospital with a history of a chronic productive cough for the previous six months, accompanied by occasional hemoptysis and the loss of twenty-five pounds, following an attack of pneumonia six months before. Examination showed a chronically ill man with a flattening of the right side of the chest and decreased breath sounds over the right side of the upper part of the chest around

the site of a completely healed scar where a drainage of a lung abscess had been performed in May, 1939. The x-ray picture showed an infiltration throughout the right upper lobe. Pus was noted coming from the right upper lobe bronchus on repeated bronchoscopies performed five times during July and August, 1939, without any permanent relief of symptoms or clearing of the pulmonary infiltrations. A cautery pneumonotomy was performed Sept. 5, 1939, followed by a chronic bronchopleurocutaneous fistula and relief of symptoms. Sulfur granules were found repeatedly from the bronchoscopic aspirations and abscess drainage, establishing a diagnosis of actinomycosis. An unsuccessful attempt at closure of the fistula was performed April 1, 1942, by means of a muscle flap. The wound broke down, and again sulfur granules were found in the drainage. The fistula finally closed spontaneously in June, 1942, and the patient had no further recurrence of his symptoms. A course of x-ray therapy was given during August, 1939.

This patient was admitted five times from 1939 to 1942.

E. DeJ (admitted March 26, 1940, discharged May 14, 1940), a 29-year-old woman, was admitted on the Chest Service of Barnes Hospital with a history of a chronic productive cough for one year before admission, accompanied by occasional hemoptysis, intermittent fever, and pain in the right side of the posterior part of the chest. She had been treated during the previous year by a right pneumothorax, sulfonamides and repeated bronchoscopic aspirations, without any permanent improvement. Examination showed a chronically, but not acutely, ill woman with râles and suppression of breath sounds over the right side of the upper part of the chest. A roentgenogram revealed a dense perihilar infiltration in the upper part of the right lung. A bronchoscopic aspiration April 2, 1940, revealed thick pus coming from the right upper lobe bronchus which showed sulfur granules on direct examination and actinomyces on culture. A two-stage cautery pneumonotomy drainage of a lung abscess in the upper right lobe was performed April 7, 1940, and May 22, 1940. The abscess cavity was packed loosely with an acriflavine gauze dressing every two days for a period of approximately three months until the cavity became completely obliterated. The patient has remained well and active until the present time without a recurrence of the cough or any other symptoms or signs of her previous infection.

A. F., a 51-year-old man, was admitted on the Chest Service with a history of a persistent productive cough with large amounts of foul sputum and fever, accompanied by pain in the right side of the chest, for the past three weeks. Examination showed a chronically and acutely ill man with dullness and suppression of breath sounds over the right side of the lower part of the chest. An x-ray picture showed an infiltration throughout the lower right lung with a homogenous opacity suggesting fluid at the periphery. An empyema cavity was drained first by closed drainage Oct. 5, 1931, followed by an open drainage with rib resection Oct. 10, 1931. A microscopic examination of the pus from the empyema showed sulfur granules, establishing a diagnosis of actinomycosis. The patient's cough and fever continued, accompanied by a persistence of the shadow in the lower right lung, suggestive of an abscess. A cautery pneumonotomy drainage was performed Oct. 27, 1931, and the wound packed with a gauze dressing every two days. A course of deep x-ray therapy was given during November and December, 1931. The draining fistula finally closed after about three months, although the patient continued to cough up moderate amounts of blood and foul sputum during the following year. He returned for a revision of the thoracotomy drainage Dec. 1, 1932, which opened up a bronchopleurocutaneous fistula and relieved him of the cough and temperature elevation. Another course of deep x-ray therapy was given at this time. The draining sinus in the chest gradually closed and his symptoms disappeared over a course of several months. He remained well for the next five years. Then, in 1937, he began to develop hypertensive cardiac disease with intermittent attacks of decompensation. He continued to have cardiac trouble until he died Oct. 16, 1940. The hypertensive heart disease had no apparent connection with the actinomycosis, which had apparently remained completely cured since the early part of 1933.

This patient was admitted Sept. 22, 1931, discharged Jan. 22, 1932, readmitted Nov. 20, 1932, and discharged Dec. 11, 1932.

J. L., a 17-year-old girl, was admitted on the Chest Service of Barnes Hospital with a history of a chronic productive cough since November, 1939, after having had a tonsillectomy under general anesthesia in August, 1939. The patient suffered from a gradually increasing fever and cough, accompanied by disorientation and mental confusion. Examination showed an acutely and chronically ill girl with dullness and absence of breath sounds over the lower left lung and roentgenograms revealed a pleural effusion and lung abscess. A bronchoscopy Feb. 7, 1940, showed large amounts of foul pus coming from the lower left lobe. A total of nine bronchoscopic aspirations failed to produce much improvement in the symptoms. A cautery pneumonotomy Feb. 21, 1940, provided drainage for both the empyema and lung abscess. Sulfur granules were found in both the empyema fluid and the bronchoscopic aspirations, establishing a diagnosis of actinomycosis. The drainage tract proved inadequate and eventually it closed, accompanied by recurrence of fever and productive cough. Another cautery pneumonotomy was performed April 10, 1940, resulting in adequate drainage of the lower left abscess, followed by an immediate reduction of fever and cough. At the time of discharge in May, 1940, a small rubber tube had been substituted for the gauze pack in the abscess cavity. The tube was discarded in less than two months and the draining fistula was allowed to close completely. A course of x-ray therapy was given in February and March, 1940. The patient has been followed up until the present time without any evidence of a recurrence of actinomycosis.

This patient was admitted Feb. 4, 1940, and discharged May 5, 1940.

R. M., a 49-year-old man, was admitted on the Chest Service of Barnes Hospital with a history of a chronic cough, low-grade fever, pain in the left chest, weakness, weight loss and malaise since June, 1942. An examination in January, 1942, revealed a chronically ill man with a diffuse pneumonitis throughout the lower left lung, which appeared nonspecific on that examination. The pneumonitis and accompanying symptoms persisted, requiring a lobectomy in June, 1943. This was followed by a postoperative empyema with a persistently draining sinus in the left side of the chest wall, accompanied by continued low-grade fever, weakness and weight loss, until he was given a course of penicillin therapy of 2,000,000 units over a two-week period in March, 1944. At that time, actinomyces were noted in the drainage from the chest wall sinus, confirming the diagnosis made by finding actinomyces on pathologic examination of the surgical specimen following lobectomy. The patient has continued to gain weight and strength during the past year without any further drainaing sinuses in the chest wall. He is now able to return to full-time employment for the first time in four years.

N. A., a 46-year-old woman, was admitted on the Chest Service at Barnes Hospital with a history of a chronic pneumonitis accompanied by a productive cough and low-grade fever since 1938. This was thought at first to be due to tuberculosis and was treated by bed rest without any permanent improvement. A lung abscess and empyema developed in 1941, which was drained by a pneumonotomy. The patient returned in October, 1942, with the same symptoms, which were thought at that time to be due to a lung abscess in the lower right lobe. This was treated by bronchoscopic aspirations, without any improvement in the cough, fever, weakness and malaise. Definite evidence of bronchiectasis of the lower right lobe was noted in June, 1943, and a lobectomy was performed with the removal of the lower right lobe. This was followed by an intermittently recurring empyema, chest wall abscesses, and draining sinuses requiring repeated thoracotomy revisions in August, 1943, October, 1943, and November, 1943. At this time, the patient became very acutely ill with nausea, vomiting, jaundice, a daily temperature of 40° C., and a very much enlarged and tender liver. Several more thoracotomy revisions were performed during this time, and finally a Schede thoracoplasty was performed with the removal of the entire lower portion of the right lateral chest wall. Repeated courses of penicillin were also given, amounting to 8,340,000 units. The temperature was temporarily reduced with each course of penicillin and the patient's general condition improved markedly with the return of her appetite. Actinomyces were finally found in the drainage from the chest wall sinuses and an exploration showed evidence of a very small

amount of pus beneath the diaphragm on the right side. The draining sinuses finally healed completely in August, 1944, and the patient has now regained seventy pounds and is able to resume her normal housework.

Summary of treatment: thoracotomy, Dec. 13, 1941; lobectomy, June 30, 1943; thoracotomy, Aug. 4, 1943, Oct. 27, 1943, and Dec. 24, 1943; Schede thoracoplasty, March 1, 1944; thoracotomy revision and drainage of subdiaphragmatic abscess, April 21, 1944; penicillin, 8,340,000 units, Nov. 15 through April 8, 1945.

A. M., a 19-year-old girl, was admitted on the Chest Service of Barnes Hospital with a history of a chronic productive cough which had grown progressively more severe since January, 1942, accompanied by a low-grade fever and weight loss. This had not been influenced by a tonsillectomy in June, 1942. When first seen here, the patient was acutely and chronically ill with multiple abscesses scattered through the lower part of the right lung. These were treated without improvement by repeated bronchoscopic aspirations in September, 1942, and then drained by a two-stage cautery pneumonotomy in September and October, 1942. The chronic cough and low-grade fever continued and a revision of the cautery pneumonotomy was performed in December, 1942, with the drainage of further lung abscesses. As this did not control the continued temperature elevation and chronic productive cough, a lobectomy was performed in March, 1943, with the removal of the right middle and right lower lobes. The operative procedure was difficult due to dense scar tissue around the old drainage site and to the replacement of a large portion of the lung by scar tissue between the lung abscesses. The postoperative course was stormy, being complicated with a bronchopleural fistula, post operative empyema, and chest wall abscesses which required drainage in May, 1943. The productive cough, low-grade fever and persistent drainage from several sinus tracts in the right side of the chest continued, accompanied by occasional severe pulmonary hemorrhages during the next eight months. Another cautery pneumonotomy drainage of a recurrent lung abscess was performed in January, 1944. Cough, fever and drainage continued, accompanied by x-ray evidence of several large areas of rarefaction in the upper right lobe, suggesting the desirability of completing the pneumonectomy by removing the remaining right upper lobe. This was attempted in March, 1944, but found impossible due to the extreme vascularity of the remaining lung tissue which was honeycombed by abscess cavities and very extensive scar tissue combining the lung and chest wall. Another cautery pneumonotomy was therefore performed in place of the proposed lobectomy. This again did not reduce the febrile reaction or productive cough until after a course of penicillin therapy, consisting of 1,986,000 units of penicillin, had been administered over a two-week period. The patient was then discharged in a relatively afebrile and much improved condition. The cough and fever soon returned, however, requiring another cautery pneumonotomy April 29, 1944. This final procedure almost completely burned out the remainder of the right lung, accomplishing a cautery pneumonectomy in reality. Another 2,000,000 units of penicillin were administered during the postoperative period and this time the patient remained afebrile without any recurrence of the productive cough and the draining sinuses gradually closed over during the following four months. Immediately after the last stage of the cautery pneumonectomy, actinomyces were found for the first time by Dr. Phillip Varney.* The patient has remained in good health without any further draining sinuses, cough or fever during the past one year. She has also gained fifty pounds and resumed her normal activities helping with a dairy farm, in addition to being married recently.

Summary of treatment: eight hospital admissions, seven bronchoscopic aspirations, five cautery pneumonotomies, three additional drainages of chest wall abscesses, one lobectomy, repeated courses of sulfadiazine and two courses of penicillin of approximately 2,000,000 units each.

DISCUSSION

Etiology.—The organism under discussion, the genus Actinomyces, belongs to the family Actinomycetaceae, of the order Actinomycetalis, of the class

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Schizomycetes. There are seventy recognized species, of which eleven are animal parasites and fifty-nine are plant parasites. A number of these species are burdened with several different names, adding further to the confusion. The most commonly found animal parasite is the *Actinomyces bovis,* which causes the familiar "lumpy jaw" of cattle. The most interesting species from our point of view is the *Actinomyces hominis,* or *Streptothrix hominis,* or Wolf-Israel type of organism which causes the typical picture of actinomycosis in human beings. Several of the other species are found in chronic lung infections, such as *Actinomyces necrophorus* in abscesses, *Actinomyces gedanensis* in chronic pneumonitis, and *Actinomyces candidus* Petruschke or *Actinomyces asteroides* Effenger in association with pulmonary tuberculosis. *Actinomyces gonadiformis* is frequently found in the tonsillar crypts.

These different organisms have certain distinguishing cultural characteristics, but all grow slowly, preferably at body temperature. A simple and yet complete analysis is presented by Kerlan.[10]

Pathogenicity.—Great difficulty has been encountered in attempting to reproduce typical actinomycotic infections in animals. After repeated trials with *Actinomyces israeli* organisms isolated from human victims, Roseberry, Epps and Clark[11] report that animal injection of virulent strains proved pathogenic in only a few cases. Infection resulted only from the intrapleural or intraperitoneal injection of the organisms. The best results were said to have been obtained from the intrapleural injection of organisms accompanied by pulverized salivary calculi.

Infections are thought to occur more frequently in those persons around cattle, suggesting a slight contagious aspect of the disease, although an epidemic, or even the infection of several members of the same family, is practically unknown.

Incidence and Distribution.—The incidence of human infection with pathogenic actinomyces is generally thought to be very low. Lord[12] reported only sixty-five cases in 122,408 admissions to the Massachusetts General Hospital up to 1925, and Miller and Merkel[13] found only one in 12,000 autopsies. The Mississippi River valley and the northwest section of the country are thought to show a higher incidence of actinomycosis than other parts of the country, according to Fiske[14] and Alexander.[15]

The jaw and cervical region is by far the most frequent site of infection in human beings, accounting for about 60 per cent of all of the infections.[7] Less than 25 per cent of the infections[1, 7] are present in the lung and chest wall.

Clinical Picture.—Pulmonary actinomycosis usually starts as a chronic pneumonitis accompanied by fever, cough and, especially, chest pain. It may continue over a period of many months as an unresolved pneumonitis, or areas of necrosis may occur resembling lung abscesses which drain themselves at least partially through the bronchial tree. These abscesses tend to be small and multiple, which make x-ray diagnosis and surgical drainage difficult. The disease may extend peripherally through the chest wall, producing a periostitis and, later, rib necrosis. A soft tissue swelling may occur on the chest wall and form

a chronically draining sinus after it is opened. A number of such draining sinuses may be present in far-advanced cases. This organism does not respect any barriers and is capable of penetrating in any direction through bone or fascial planes, or into the mediastinum or the spinal canal, resulting in a rapidly fatal meningitis. Transmission from above downward through the diaphragm is possible but, as in other types of infection, the extension is usually from the peritoneal to the pleural cavity. A blood-borne dissemination in the liver, lungs and brain is rapidly fatal.

Diagnosis.—The most satisfactory method of making a diagnosis of actinomycosis is by finding the organisms in a fixed section of tissue taken from around a draining sinus tract. Sometimes the typical sulfur granules can be seen also and a diagnosis made by direct microscopic examination of the pus draining from such a sinus tract, or occasionally from the sputum or bronchoscopic aspirations of the bronchial secretion. Aspiration of an unruptured abscess furnishes more satisfactory material for examination than does drainage from a chronic sinus contaminated by secondary infection. The fungi can be grown also on culture media incubated at body temperature over periods of several weeks. The more pathogenic varieties show anaerobic tendencies and grow very slowly, according to Varney, who has isolated the fungi on culture media in several of the cases reported. The difficulty in isolating actinomycotic organisms during the early stages of many cases is demonstrated in some of the foregoing reports. Repeated routine examinations of the sputum had been made over a period of from two to three years in certain instances without finding the organisms until special efforts were made in connection with penicillin research. It is for this reason that I believe an appreciable number of early cases are not definitely diagnosed by routine methods.

Treatment.—It is noteworthy that in all of the patients reported on in this series, as well as in most of the successfully treated patients whose cases were reviewed, surgical drainage or excision represented the basic form of treatment. This was supplemented in many cases by other forms of therapy, such as x-ray, sulfonamides, penicillin, thymol and potassium iodide, with varying degrees of credit being given to different agents. The popularity of potassium iodide in the treatment of actinomycosis in which the Wolf-Israel organism is the causative agent has been gradually decreasing during the past fifteen years. The beneficial effect of iodides on *A. bovis* infections in cattle apparently is not present when used against *A. hominis* organisms.[16] Although x-ray therapy is recognized as contributing to a number of cures of cervical actinomycotic infections, its effectiveness in the deep-seated thoracic infection is extremely questionable. Only one in all of the reported cures resulted from x-ray therapy alone. Several favorable reports of the use of sulfonamides, especially in association with surgical drainage, were mentioned previously. Penicillin will perhaps prove even more effective than the sulfonamides in controlling the severe toxic symptoms present in extensive actinomycosis between stages of surgical drainage. The abscesses in this disease tend to be so numerous and widespread that a single drainage procedure frequently is inadequate to eliminate all of them at one time.

In considering the results of any form of therapy, it should be emphasized that actinomycosis is a disease which is often characterized by long latent periods, followed by recrudescence of symptoms. One should hesitate, therefore, to speak of cured cases until after the lapse of at least several years. As in tuberculosis, it is probably wiser to designate apparent cures as arrested cases.

CONCLUSION

1. Cures of primary thoracic actinomycosis are possible.

2. Repeated extensive surgical drainages, débridements or excisions have effected the greatest number of cures.

3. Internal medication and x-ray therapy should not be allowed to postpone surgical drainage of infected areas, although they may be used effectively to supplement the drainage procedures.

4. Penicillin appears to offer considerable promise in alleviating the severe toxic symptoms when used in conjunction with surgical drainage.

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PRODUCTION OF MUCUS IN PRIMARY NEOPLASMS OF THE LUNG*

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IT IS a well-known fact that certain adenocarcinomas secrete mucus. Among those carcinomas which commonly secrete mucus are carcinomas of the stomach, colon, and rectum and, less frequently, carcinomas of the gall bladder, pancreas, prostate gland, and breast.

Pilcher[1] in studying the significance of the production of mucus in carcinoma of the prostate gland and Ochsenhirt[2] in a similar study of carcinomas of the large intestine and rectum concluded, independently, that the more malignant or less differentiated the carcinoma, the less numerous the cells secreting mucus and the lower the degree of malignancy the greater the production of mucus.

Bell,[3] Horsley,[4] Illingworth and Dick,[5] and Cutler and Buschke[6] have written of the production of mucus in carcinoma of the breast. According to Cutler and Buschke[6] there is evidence to indicate that this form of mammary carcinoma is less malignant and grows more slowly than other forms and metastasizes late in its course.

Boyd,[7] Cutler and Buschke,[6] and Illingworth and Dick[5] have written concerning production of mucus in gastric carcinoma. Illingworth and Dick[5] stated that mucus is present in the metastatic lesions. Mucus has been found in carcinoma of the gall bladder according to Illingworth and Dick,[5] Kaufmann,[8] and Cutler and Buschke.[6] Cutler and Buschke[6] and McFarland[9] also stated that gelatinous carcinoma of the pancreas is encountered.

Little attention has been paid to the production of mucus in epithelial neoplasms of the lung. Olson[10] from a study of primary carcinoma of the lung, based on sixty-nine post-mortem examinations, stated that the chief distinguishing features of adenocarcinoma of the lung are the secretion of mucus and the resemblance to the bronchial mucous glands. He found mucinous secretion in the metastatic lesions.

Our study was initiated because carcinoma of the lung has been seen occasionally in which the production of mucus has been great. In such cases, the question was raised whether the lesion in the lung was primary or metastatic. It was deemed important, therefore, to determine how much mucus can be produced by a primary carcinoma of the lung.

Fifty-three epithelial neoplasms of the lung which had been removed by either lobectomy or pneumonectomy were studied. Sections from each neo-

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plasm were stained with hematoxylin and eosin and Mayer's mucicarmine stains. It was felt that the neoplasms in this group were primary in the lung since other primary lesions were not evident at clinical examination. It is realized that this does not furnish absolute proof that the carcinoma was primary in the lung. To have absolute proof it would be necessary to examine all the organs thoroughly at necropsy.

Forty-four of the fifty-three neoplasms were bronchiogenic carcinomas and nine were lesions of the adenoma type. We are using the term "bronchiogenic carcinoma" not of necessity to indicate an origin from bronchial epithelium but rather as a term which is commonly employed in the literature to distinguish primary carcinoma of the lung from the less rapidly growing neoplasms included in the adenoma group. The bronchiogenic carcinomas were classified as squamous-cell carcinomas, adenocarcinomas, and those in which differentiation into either of the preceding two groups was not possible. The adenoma group of neoplasms consisted of eight adenomas and one cylindroma. The terms "adenoma" and "cylindroma" are employed because they are commonly recognized in the literature. Although adenoma and cylindroma of the lung present a different histologic pattern, they apparently pursue a similar clinical course and present a similar gross appearance.

When mucus was present in any of the neoplasms studied, the quantity present was graded 1, 2, 3, or 4, the more mucus present the higher the grade. Generally when the mucus produced by the neoplastic cells was confined to the cells, it was graded 1 or 2; when it was both intracellular and extracellular, it was graded 3 or 4.

RESULTS

Bronchiogenic Carcinoma.—By utilizing the hematoxylin and eosin stain, twenty of the forty-four bronchiogenic carcinomas were diagnosed as squamous-cell carcinoma; fourteen as adenocarcinoma, one of which was a papillary adenocarcinoma; two were a mixed type of adenocarcinoma and squamous-cell carcinoma; in eight it was impossible to determine the type of carcinoma. It is a commonly accepted fact that although every adenocarcinoma does not necessarily produce mucus, every carcinoma which produces mucus is evidence per se that the cancer cells are glandular in nature. With the aid of the mucicarmine stain, it was impossible to find mucus in any of the squamous-cell carcinomas. In some sections, the keratin of the individual cells stained pale pink with mucicarmine but the intensity of this reaction was not so bright as that which is given by mucus. Mucus was formed in both of the lesions diagnosed as mixed squamous-cell carcinoma and adenocarcinoma (Fig. 1). Sections from twelve of the fourteen adenocarcinomas stained with hematoxylin and eosin revealed mucus (Fig. 2, *a*). In three of the eight cases in which the type of carcinoma could not be classified from sections stained with hematoxylin and eosin, staining with mucicarmine revealed mucus (Fig. 2, *b*). When there was spread of adenocarcinoma to the regional nodes, sections of the involved nodes stained with mucicarmine also revealed mucus.

The final classification of the forty-four bronchiogenic carcinomas after examination of sections stained with the hematoxylin and eosin and those stained

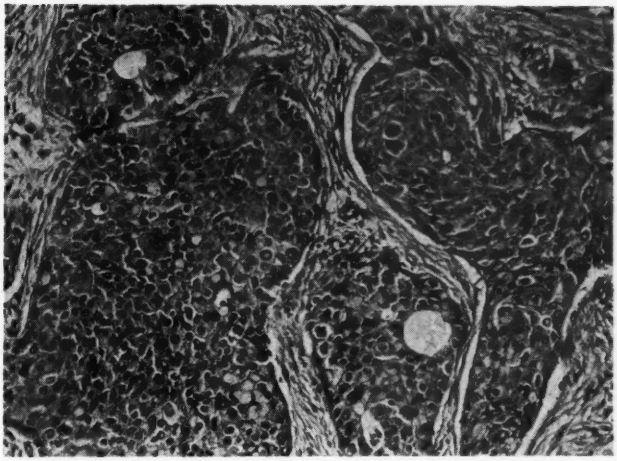

Fig. 1.—Primary carcinoma of the bronchus; mixed squamous-cell carcinoma and adenocarcinoma. The groups of carcinoma cells with slight keratinization, and the formation of mucus in several areas may be noted (×115).

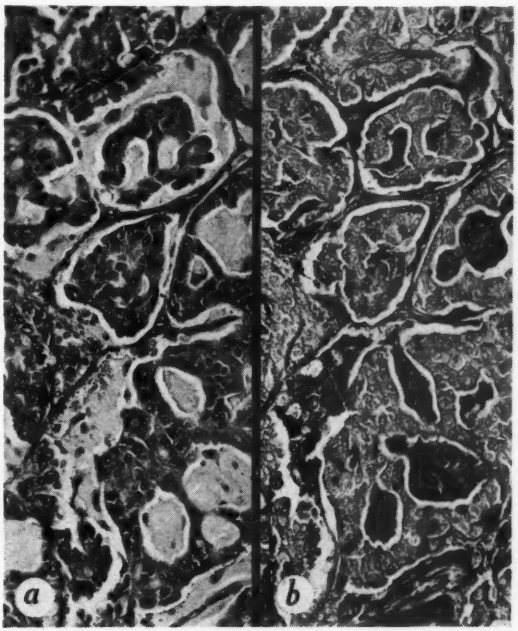

Fig. 2.—Adenocarcinoma of bronchus with production of mucus, *a*, stained hematoxylin and eosin (×120); *b*, stained mucicarmine (×120).

with mucicarmine stains revealed twenty squamous-cell carcinomas, seventeen adenocarcinomas, two mixed type of squamous-cell carcinomas and adenocarcinomas, and five undifferentiated carcinomas which were considered of indeterminate type.

In five of the adenocarcinomas, the amount of mucus which was produced was graded 1. In five, it was graded 2; in one it was graded 3, and in four it was graded 4. In the two mixed squamous-cell carcinomas and adenocarcinomas it was graded 2. The four lesions with grade 4 mucus are examples of bronchiogenic carcinomas which would ordinarily be considered metastatic from some other organ. However, as has been stated, there was no evidence of another primary lesion and they must be considered as primary in the lung. The incidence of metastasis to regional nodes was much higher in the patients with adenocarcinoma than in those with squamous-cell carcinoma. From such a small series of cases, however, it is impossible to draw conclusions concerning the correlation of production of mucus in bronchiogenic carcinoma with the incidence of metastasis or prognosis.

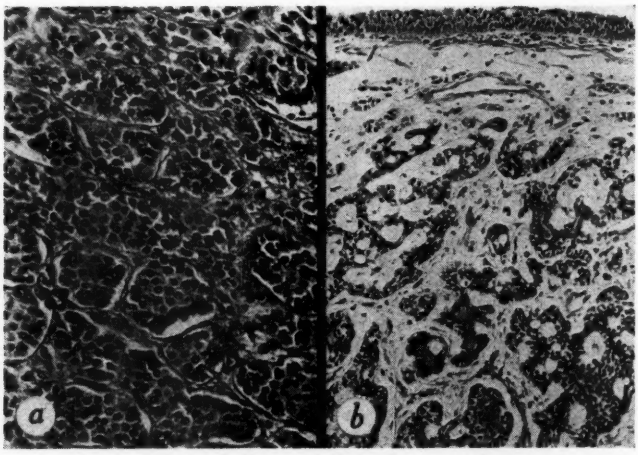

Fig. 3.—*a*, Adenoma of the bronchus; the lack of secretion may be noted (×200); *b*, cylindroma of the bronchus; there is definite secretion of mucus (hematoxylin and eosin ×85).

Adenoma Group.—Of the nine tumors belonging to the adenoma group, eight were adenomas and one was a cylindroma. There was no mucus produced in any of the adenomas (Fig. 3, *a*). The one cylindroma produced mucus of grade 3. The staining reaction for mucus in this cylindroma was not as intense as that of the mucus of adenocarcinoma (Fig. 3, *b*).

SUMMARY

Production of mucus comparable in amounts to that produced in certain carcinomas of the gastrointestinal tract occasionally will be found in primary carcinomas of the lung. Mucus also is produced in the nodal metastatic lesions

from primary adenocarcinoma of the lungs. Adenomas of the lung do not produce mucus. On the other hand, cylindromas of the lung produce mucus in appreciable quantities.

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LIPOMA OF THE BRONCHUS: DISCUSSION OF BENIGN NEOPLASMS AND REPORT OF A CASE OF ENDOBRONCHIAL LIPOMA

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BENIGN bronchial neoplasms comprise a group of tumors which though histologically benign may, through occlusion of a bronchus, give rise to atelectasis and often fatal suppuration in the tissue of the lung distal to the occlusion. Formerly these tumors were discovered only at necropsy and were considered to be medical curiosities. Now by means of bronchoscopy, thoracic surgery, and routine roentgenograms of the thorax, these tumors are being diagnosed clinically. A review of the literature regarding these tumors reveals several interesting facts. First, the pathogenesis and growth potentialities of certain of these tumors are in dispute. Second, fat is a frequent component of benign bronchial neoplasms.

REVIEW OF THE LITERATURE

As early as 1854 Rokitansky[1] found at necropsy a submucous bronchial lipoma which almost filled the lumen of a left main bronchus. It is impossible, however, because of insufficient microscopic data, to tell whether this was a true lipoma or a hamartoma. In 1879 Laboulbène[2] mentioned a lipoma of the bronchus containing spindle-shaped cells. Again it is not certain whether this was a true lipoma, as the spindle cells were not described in detail. In 1927 Kernan[3] reported the bronchoscopic removal of a submucous lipoma from the bifurcation of the trachea. The tumor consisted of mature fat cells supported by a vascular fibrous tissue and was covered with bronchial mucous membrane. Myerson[4] in 1928 bronchoscopically removed a fibrolipoma obstructing a left main bronchus, but did not describe the microscopic appearance of the tumor. Wessler and Rabin[5] in 1932 mentioned seeing a pedunculated fibrolipoma in a left lower lobe bronchus. No histologic description of the tumor was given. Jackson and Jackson[6] in 1932 listed a number of benign tumors which they had encountered bronchoscopically. Among these were a lipoma and a fibrolipoma. However, they gave no microscopic description and did not state whether the tumors occurred in the trachea or bronchi. In 1939 McGlade[7] removed a polypoid fibrolipoma occluding a left main bronchus. The tumor, which was composed of fibrous tissue and fat, was removed through the bronchoscope. Vinson and Pembleton[8] in 1942 removed a pedunculated lipoma from the left main bronchus through the bronchoscope. Microscopically the tumor consisted of fatty tissue covered with normal bronchial epithelium.

The following is the report of a case in which an endobronchial lipoma was found at operation. Pathologic examination of tissue from the tumor was made.

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REPORT OF CASE

A white man, 51 years old, registered at the Mayo Clinic Aug. 6, 1945. He first consulted his local physician in July, 1943, because of fatigue, loss of weight, frequent colds and a productive cough. A diagnosis of pulmonary tuberculosis was made from the roentgenograms of the thorax. Tubercle bacilli could not be found in the sputum. Despite the admonitions of his physician he continued to work. The cough grew worse, and he often expectorated as much as 5 ounces (150 c.c.) of yellow, occasionally blood-streaked sputum a day. Night sweats and a temperature of from 99 to 101° F. developed. In April, 1944, the patient returned to his local physician. A roentgenogram of the thorax revealed "extensive

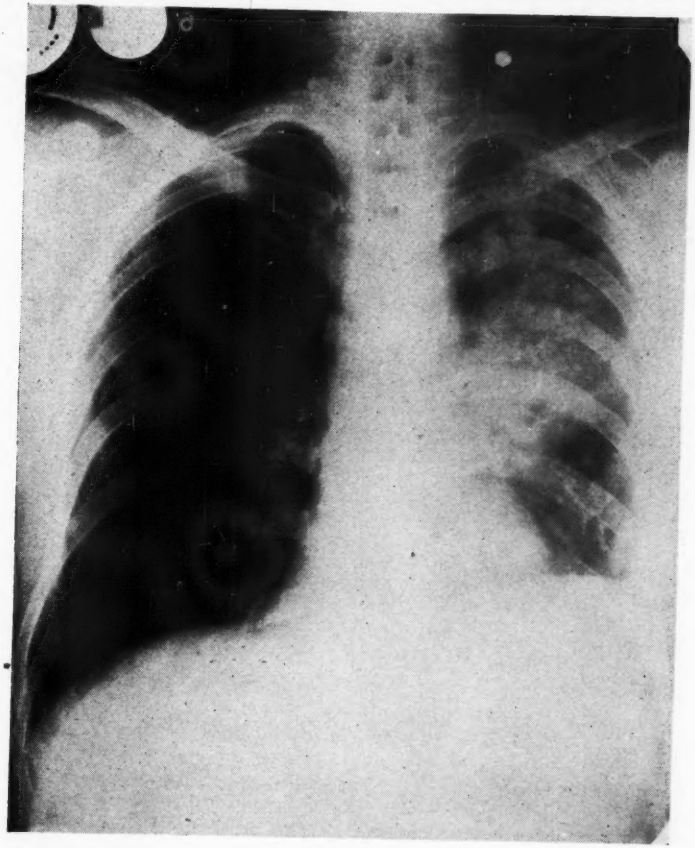

Fig. 1.—Atelectasis of the upper lobe of the left lung.

infiltration of the left upper lobe" which was interpreted as pulmonary tuberculosis. Examination of sputum again revealed no tubercle bacilli. He was sent to a tuberculosis sanatorium where he spent one year in bed. Left artificial pneumothorax was created and the left portion of the diaphragm was paralyzed by crushing the left phrenic nerve. Although he gained weight, productive cough, fever and night sweats continued unabated. Repeated examinations of the sputum failed to reveal tubercle bacilli, fungi or other pathogens. His medical attendants suspected tumor of the lung and referred the patient to the clinic.

Physical examination at the clinic in August, 1945, revealed a well-nourished man with a temperature of 100° F. The blood pressure was 150 mm. of mercury systolic and 86 mm. diastolic, and the pulse rate was 112 beats per minute. There were dullness to percussion and a decrease in the breath sounds and tactile fremitus over the left upper part of the thorax. The remainder of the physical examination gave negative results.

Examination of the urine gave negative results. Examination of the blood revealed the following: hemoglobin, 12.5 Gm. per 100 c.c.; erythrocytes, 4,690,000 per cubic millimeter; leucocytes, 22,600 per cubic millimeter; and sedimentation rate, 50 mm. per hour (Westergren method). The differential white cell count was not pathognomonic. Repeated examinations of the sputum failed to reveal tubercle bacilli, fungi or other pathogens.

Fig. 2.—Lipoma in the main bronchus of the upper lobe of the left lung.

Roentgenographic examination of the thorax revealed a granular process involving the upper lobe of the left lung and a poorly defined large left hilar mass. The heart and mediastinum were shifted to the left (Fig. 1).

Bronchograms were made, but the upper lobe of the left lung could not be visualized. The bronchi of the lower lobe of the left lung appeared to be normal.

At bronchoscopic examination a thick purulent secretion was found in both main bronchi, but no obstructing lesion could be seen. Neither tubercle bacilli nor fungi could be found in the aspirated purulent material.

Although bronchostenosis secondary to a neoplasm, Boeck's sarcoid, fungus infection and tuberculosis were seriously considered, a definite diagnosis could not be made. Because the lesion seemed to be confined to the upper lobe of the left lung, it was decided to perform an exploratory thoracotomy in order to ascertain the nature of the process.

Accordingly, Aug. 20, 1945, the patient was admitted to the hospital, and the left portion of the thorax was explored through a left posterolateral incision. The upper lobe of the left lung was atelectatic and was attached to the parietal pleura by numerous adhesions. Many large indurated lymph nodes were present at the hilus. It was impossible to determine the nature of the lesion, and since the lower lobe of the left lung appeared normal, lobectomy was performed, the upper lobe being removed.

The patient's postoperative course was uneventful and he was dismissed from the hospital on the fourteenth postoperative day. When the patient was dismissed from the clinic, seventeen days following lobectomy, his general condition was excellent, the incision had completely healed, and no cough or fever was present.

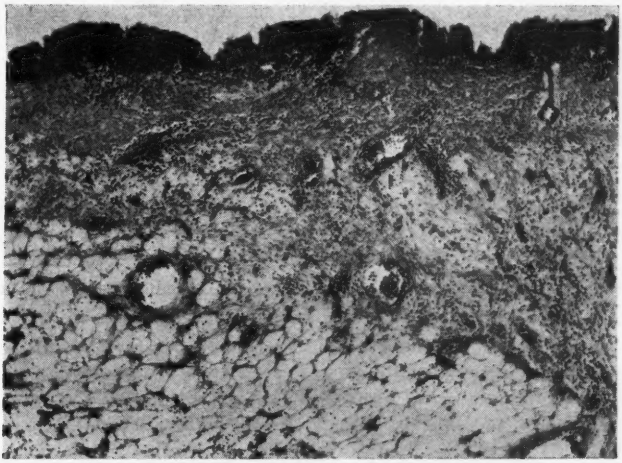

Fig. 3.—Section of the periphery of the lipoma (hematoxylin and eosin, ×50).

Pathologic Examination.—Examination of the removed lobe by the surgical pathologist revealed a soft, gray, pedunculated tumor, 2 cm. in diameter, in the main bronchus of the upper lobe of the left lung. It was attached to the wall by a narrow pedicle and completely occluded the lumen (Fig. 2). The cut surface had the appearance of a lipoma.

Microscopically the entire tumor consisted of lobules of mature fat cells supported by a delicate fibrous stroma (Fig. 3). Several regions of hemorrhage were present near the periphery. The pedunculated surface was covered with three layers of tissue (Fig. 4). The normal bronchial muscular coat was continuous with a thin layer of smooth muscle and elastic fibers surrounding the fat lobules. The lamina propria was also continued around the tumor. It was packed with lymphocytes, plasma cells, large mononuclear cells, erythrocytes and an occasional polymorphonuclear leucocyte. The outermost layer consisted of a pseudostratified columnar epithelium, continuous with that of the bronchus. At the base of the pedicle, and for 1 cm. above and below its attachment, fat lobules filled the entire submucosa from the muscular layer to the cartilage plates. There was no evidence of cartilage, bone, muscle or epithelial elements in any portion of the tumor proper.

The lung itself was atelectatic. The cut surface revealed diffuse consolidation and severe bronchiectasis. The terminal bronchi were represented by large cylindrical cavities filled with a puttylike exudate (Fig. 5).

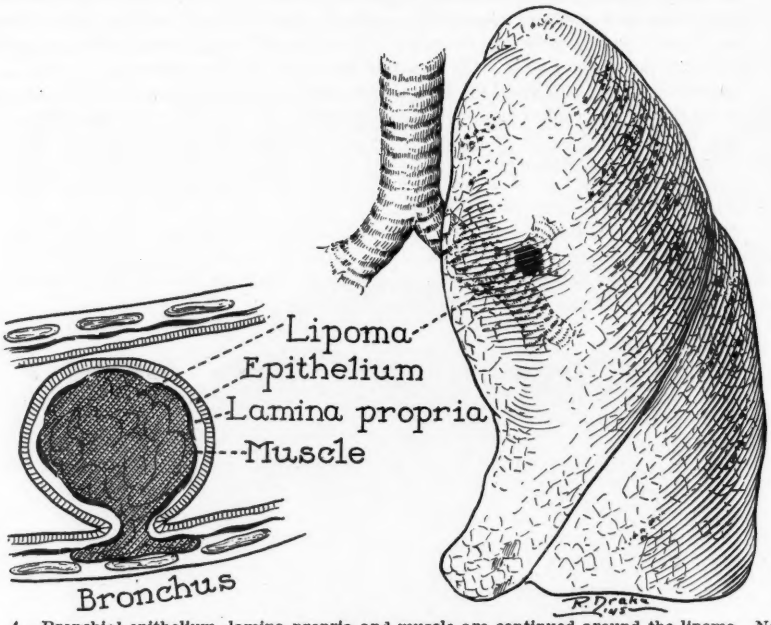

Fig. 4.—Bronchial epithelium, lamina propria and muscle are continued around the lipoma. Note fat in the submucosa.

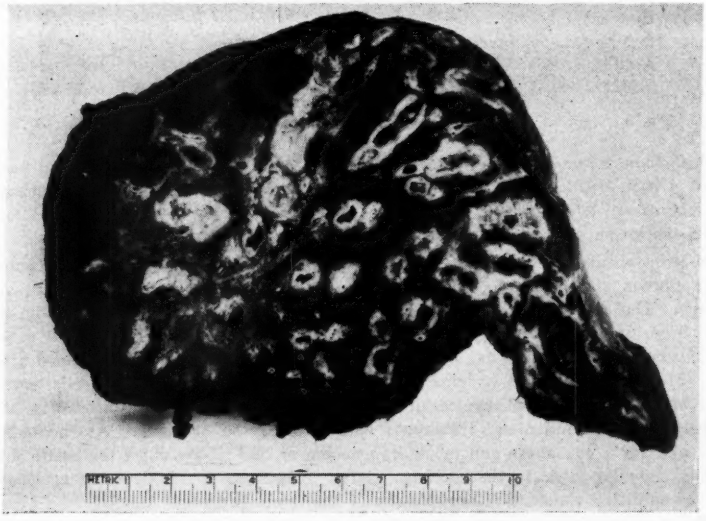

Fig. 5.—Cut surface of the lung. Note the diffuse atelectasis and severe bronchiectasis.

Microscopically the bronchi were filled with pus, and the walls and surrounding tissues were infiltrated with myriads of polymorphonuclear leucocytes, large mononuclear cells, lymphocytes and a few eosinophiles. The mucosa was atrophic and there was extensive destruction of the muscle, elastic tissue and glands. In some regions the bronchial walls were completely destroyed and replaced by fibrous tissue. Between the bronchi the lung tissue was atelectatic and was infiltrated with chronic inflammatory cells and showed diffuse fibrosis.

STUDY OF FAT IN THE BRONCHUS

The occurrence of fat in tumors of the lung is indeed an interesting phenomenon. According to Hickey and Simpson,[9] fat had been present in one-half of the reported cases of chondroma. Sixty per cent of the twenty-three hamartomas reported by McDonald, Harrington and Clagett[10] contained adipose tissue. The endobronchial fibrolipomas which were reported by Myerson, Wessler and Rabin, Jackson and Jackson, and McGlade, all contained varying proportions of fat. Recently we found a small amount of adipose tissue in an adenoma of the bronchus. Fat in adenomas has also been observed by others.[11] In addition, lipomas have been found occasionally in the trachea[12] and in the parenchyma of the lung,[13] as well as in the bronchi.

Various hypotheses have been postulated to explain the presence of fat in the tumors considered in the preceding paragraph. Chiari[14] stated the opinion that the fat which is present in enchondromas could be an extension of the fat which sometimes occurs in the submucosa of large bronchi. Hart[15] stated that fat might be present as the result of metaplasia of the connective tissue or from germinal fatty tissue "anlagen." McGlumphy[16] suggested that embryonic tissue destined to become fat might be carried from the mediastinum by the laterally growing nerves, vessels and bronchi. In 1938, Womack and Graham[11] reviewed Flint's work on the embryology of the lung and suggested that many tumors of the lung might result from failure of the embryonic bronchial buds to develop into normal structures. Since the bronchial buds originally consist of mesoderm and entoderm, this hypothesis would explain the occurrence of certain growths in which both mesothelial and entodermal elements are present, as well as those in which either the mesothelial or epithelial elements predominate. If this hypothesis concerning tumors of the lung is true, then the fat would arise from embryonic mesoderm.

Because of the frequent presence of fat in tumors of the bronchus it was decided to study human bronchi in an attempt to answer three questions: (1) Is fat a normal component of the bronchus? (2) If it is, in what portion of the bronchial wall does fat occur? (3) In which of the bronchi is fat present?

Material.—Blocks of tissue were cut from the bronchi and uninvolved parenchyma of fifteen lungs that had been surgically removed from patients who had bronchogenic carcinoma. The tissues were fixed in a 10 per cent solution of formalin, and frozen sections were cut and stained with Herxheimer's scarlet red solution. When this method is used, fat is stained orange to red. In addition, routine sections stained with hematoxylin and eosin from the lungs of twenty-five patients who died of brain tumors were examined.

Observations.—In the large bronchi, fat always was present in the fibrous tissue external to the cartilage plates. Lesser amounts were found with equal

frequency in the connective tissue (submucosa) between the cartilage and the muscular layer (Fig. 6). In this latter position, fat was most frequently found near the cartilage plates; less often it approached the muscular layer. Sometimes fat cells were seen close to the groups of glands, and, in two bronchi, fat

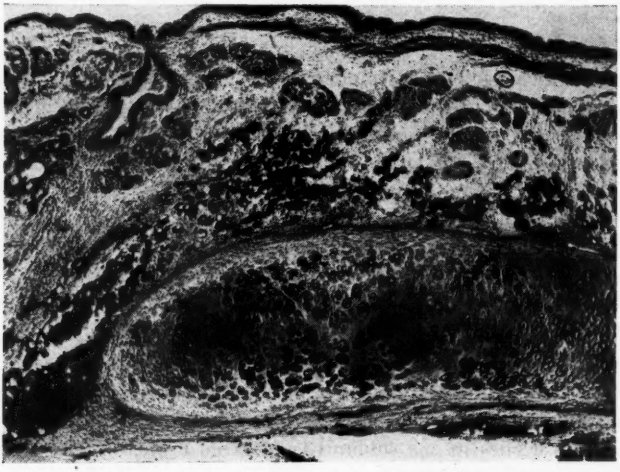

Fig. 6.—Section from the primary bronchus. Note the fat (black) in the submucosa and external to the cartilage plate (Herxheimer's scarlet red solution, ×25).

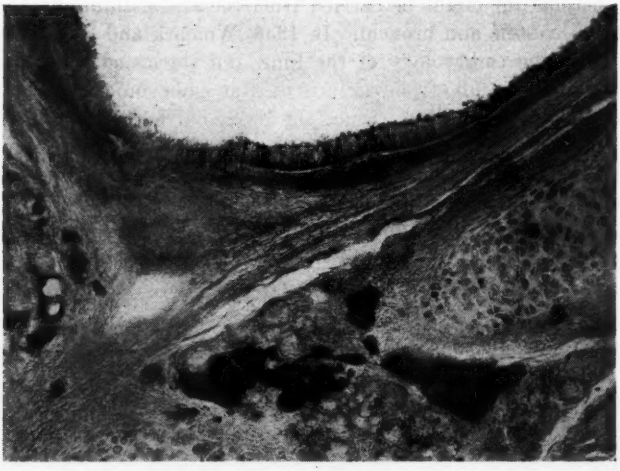

Fig. 7.—Section from the bronchus, 2 mm. in diameter. Note the fat (black) about the group of glands (Herxheimer's scarlet red solution, ×60).

cells were completely surrounded by glands. At times, clusters of fat were seen about the vessels. When little or no fat could be found in the submucosa above the cartilage it could usually be found between the cartilage plates. When the cartilages had undergone ossification, marrow cavities which contained fat

cells sometimes were found. Fat was not observed in the lamina propria of any of the bronchi examined.

In the smaller bronchi, fat was usually present in the walls in gradually decreasing amounts until the cartilages disappeared. We were unable to find fat in any portion of the bronchial tree which did not contain cartilage. As in the larger bronchi, fat was most abundant in the fibrous tissue external to the cartilage. In the submucosa it was usually found adjacent to the inner aspect of the cartilage plates and about the bronchial glands (Fig. 7).

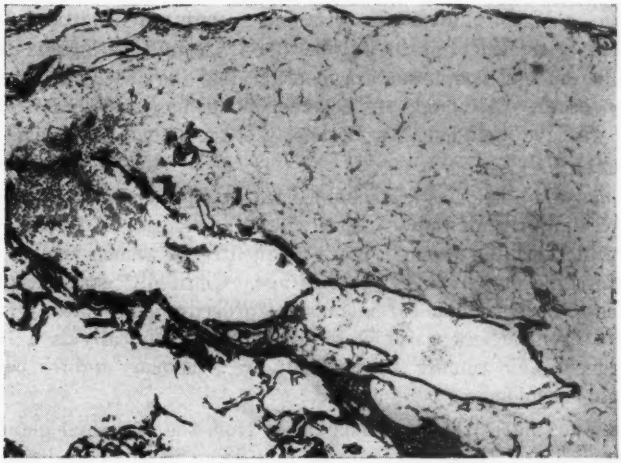

Fig. 8.—Section from a small subpleural lipoma (hematoxylin and eosin, ×30).

Comment.—Since fat is a normal component of the bronchial wall, it is not necessary to rely on a hypothesis about development or metaplasia to explain the origin of lipomas of the major bronchi. They arise from the adipose tissue normally present in the submucosa. This fact lends further support to the application of Albrecht's[17] hypothesis of hamartomas of the liver to those tumors of the lung and bronchi (hamartomas) that consist of both epithelial and mesodermal elements and whose histologic elements are all normal components of the bronchi.

The finding of adipose tissue surrounding, and even in the center of, the groups of bronchial glands might explain the occasional presence of small amounts of fat in adenomas.

Lipomas which occur in the parenchyma of the lung or beneath the pleura could arise from the fat which is often present in the smaller bronchi until they reach the diameter of 1 mm. We have seen a small subpleural lipoma but were unable to prove that it arose from a bronchus (Fig. 8).

It is difficult to explain the presence of fat in the marrow cavities in small regions of ossification of the bronchial cartilages if it is assumed that the ossification occurs after birth. It seems unlikely that mature fat cells are capable

of migrating from the surrounding tissues into the marrow cavities. A process of metaplasia is more plausible.

BENIGN BRONCHIAL NEOPLASMS OTHER THAN LIPOMAS

Benign bronchial neoplasms present perplexing problems to both the clinician and the pathologist. The clinician's diagnostic acumen is often severely taxed by the bizarre symptoms and signs produced by the accompanying bronchostenosis and its dire sequelae, atelectasis, recurrent pneumonia, bronchiectasis, pulmonary abscess and even empyema. The pathologist must settle many problems concerning pathogenesis and growth potentialities of certain of these tumors if treatment is to be placed on a sound pathologic foundation. Benign endobronchial neoplasms are mentioned in the order of frequency with which they occur. Although we consider adenomas and cylindromas to be malignant, we have included them because there are some who consider them to be benign.

Adenoma.—If adenomas are classified with benign endobronchial neoplasms, then the vast majority of these tumors will be adenomas. Adenomas almost always occur in the large bronchi, more frequently in women than in men, and usually before the patient is 40 years of age. Churchill[18] stated that the ratio of adenomas to bronchogenic carcinomas not proved microscopically is 1:20, and the ratio to bronchogenic carcinoma proved microscopically is 1:10. Of resectable bronchogenic tumors, 25 per cent are adenomas and 75 per cent are bronchogenic carcinomas.

Most writers agree that adenomas arise from the bronchial glands or their ducts. Womack and Graham suggested that adenomas be called mixed tumors because of the occasional presence of fat, cartilage, bone and muscle and suggested developmental origin. Stout[19] recalled Hamperl's descriptions of large acidophilic cells (onkocytes) in two cases and examined sections of tissue from human bronchi after they had been stained with Masson's trichrome stain. He found cells with acidophilic granules in the glands and their ducts but not in the surface epithelium. He wondered if these acidophilic cells could be the stem cells of adenomas.

Grossly, adenomas are usually smooth, rounded, pink or gray tumors attached to the bronchus by a broad pedicle.

Microscopically, the tumors are covered with a layer of epithelium which often has undergone squamous metaplasia. Between the tumor proper and the epithelium is a layer of fibrous tissue which at times is edematous and inflamed. This often leads to an erroneous diagnosis of "inflammatory tissue" if the bronchoscopist's forceps do not penetrate deeply enough into the growth. The tumor itself consists of small, regular, round or polygonal cells with a scanty cytoplasm and small, round, often darkly staining nuclei. Mitosis is rarely seen. The cells attempt to form acini but rarely succeed. The result is cords and strands of cells or a poorly formed alveolar arrangement. Bremer[20] has called attention to the striking resemblance of adenomas to fetal lung. Hamperl[21] noted the similarity of bronchial adenomas to carcinoids of the gastrointestinal

tract. In six of Hamperl's seven cases, the tumor cells had an acidophilic cytoplasm. In two cases he found large acidophilic cells which he called onkocytes. The stroma of the tumor at times may be so packed with thin-walled blood spaces lined with endothelial cells that an angiomatous nature is suggested. Womack and Graham have called attention, as previously mentioned, to the occasional presence of smooth muscle, fat, cartilage and bone in the stroma. Recently we observed fat in the stroma in one of our cases of bronchial adenoma.

Adenomas are very slow-growing tumors, but histologically they are locally invasive. They grow both into the lumen and into the wall of the bronchus. In thirteen of fifteen of the cases reported by Moersch, Tinney and McDonald,[22] the tumor had extended beyond the cartilage plates. In 90 per cent of Brunn and Goldman's[23] cases an extrabronchial extension was present and was frequently larger than the endobronchial portion. Extension to regional nodes and distant metastasis are uncommon but do occur.[11, 19, 22, 24-26] We think that bronchial adenomas should be considered as extremely slowly growing malignant neoplasms. Some observers, however, do not agree.[27, 28]

Cylindromas.—These tumors have been separated from the adenoma group by some observers because of their resemblance to the cylindromas of the salivary glands. Moersch, Tinney and McDonald found that six (13.6 per cent) of their forty-four adenomas were of the cylindroma type. Microscopically, the tumor cells form tubules filled with a mucoid secretion. A cylindroid pattern is found in which there are cylinders of tumor cells as well as cylinders of mucinous or hyaline secretion. In some cases, the stroma appears myxomatous or precartilaginous like that of mixed tumors of the salivary glands. Some observers feel that cylindromas are more infiltrative than adenomas.[22] That cylindromas can metastasize is well illustrated by the case reported by Laff and Neubuerger[29] in which a cylindroma of the right main bronchus not only invaded the adjacent pulmonary tissue and lymphatics, but metastasized to the opposite lung. Tissue from the tumor had been obtained for biopsy through the bronchoscope eleven years before death of the patient and the diagnosis was "adenomatous hyperplasia of the mucous glands, possibly carcinoma." Cylindromas occurring about the head and neck are locally invasive, metastasis is not uncommon, and recurrence following surgical extirpation is frequent. It is not at all unlikely that future investigators will prove cylindromas of the bronchus to be more malignant than adenomas. However, this is speculation; the final answer must await further study.

Fibromas.—Fibromas constituted about 9 per cent of the benign bronchial tumors reviewed by Lindgren.[30] A number of observers have reported pure endobronchial fibromas, although frequently these tumors contain other elements such as fat, muscle and angiomatous tissue.[11] Malignant change in fibromas is exceedingly rare.

Chondroma, Osteoma, Hamartoma Group.—Pure chondromas are extremely rare. In 1939 Lindgren found only nine reported cases in the literature. Pure osteomas, if they occur at all, are even less common. Most of the chondromas of which reports have been written contained fibrous tissue, fat and epithelial elements in varying proportions.[11]

Chiari[31] described two cases in which he found pedunculated, walnut-sized tumors in bronchiectatic cavities at necropsy. On microscopic examination both tumors were observed to contain cartilage, fat and glandular structures. He called these lipochondro-adenomas. Siegert[32] described a tumor which he called a chondro-angioma. It completely filled the lumen of a branch of the right main bronchus. It consisted of connective tissue, epithelial structures, cartilage and many cavernous blood spaces. Alstead[33] found a pedunculated polyp in a right main bronchus at necropsy. It consisted of fat, fibrous tissue, smooth muscle and epithelial elements. Honig[34] described a smooth, ovoid tumor arising from the left main bronchus. It was growing down into the pulmonary parenchyma. On histologic examination it was observed to contain fat, bronchial mucosa, smooth muscle and cartilage. Honig classified this as a hamartoma. Gebauer[35] removed a hard polypoid tumor, 8 mm. in diameter, from the bronchus of the lower lobe of the right lung through the bronchoscope. On microscopic examination it was observed to contain cartilage, bone, mucoid connective tissue and fat. He called this an osteochondroma. Albrecht in 1904 introduced the term hamartoma in describing certain tumors of the liver. He defined hamartomas as those tumors containing the normal components of an organ in an abnormal quantity, arrangement and degree of differentiation. In our opinion, the cases reported by Chiari, Siegert, Alstead, Honig and Gebauer are hamartomas, since all of the elements present in these tumors are normal components of the bronchi. We agree with Feller[36] and Ribbert,[37] who stated that most enchondromas are really hamartomas, as careful examination will usually reveal the presence of other elements in addition to cartilage. However, it cannot be denied that true chondromas, as described by Davidson[38] and others, occasionally occur. Malignant transformation is exceedingly uncommon. However, it is possible that the osteoid chondrosarcoma reported by Greenspan[39] may have arisen from a hamartoma or chondroma.

Lipoma.—Lipomas have already been discussed. We need only emphasize that fat occurs in association with other tumors far more often than it occurs alone.

Miscellaneous Tumors.—Papillomas have been reported to occur in the bronchi[40] but are more frequently found in the trachea, where, according to Graham, Singer and Ballon,[41] they are the commonest benign tumor. Although muscle is sometimes found in association with other tumors it is very rarely the only constituent. Kramer[42] reported in 1939 that he found a pedunculated myoblastoma in a bronchus of the lower lobe of the right lung. Myoblastomas are generally benign but may be malignant. Kramer explained the presence of a tumor arising from striated muscle in a bronchus by pointing out that the bronchial anlagen arise from the foregut, which contains striated muscle. Angiomas have been reported but, as emphasized by Womack and Graham and Stout, adenomas may be exceedingly vascular, and it is not unlikely that many have been mistaken for angiomas. Pollack, Cohen and Gnassi[43] could find only one reported case of lymphoma in 1938. Radestock[44] has reported aberrant colloid thyroid tissue obstructing a right main bronchus.

Treatment.—The treatment of fibromas, chondromas, hamartomas and lipomas is the same. The procedure of choice is removal with bronchoscopic forceps, surgical diathermy and local implantation of radon seeds, if necessary. Removal of pulmonary tissue need be done only when the tumor cannot be successfully treated through the bronchoscope, or when suppuration in the pulmonary tissue distal to the tumor endangers the health of the patient. On the other hand, there is much disagreement concerning the proper treatment of the adenoma-cylindroma group. Those who consider these tumors strictly benign[27] treat them just as they would the other benign tumors mentioned previously in this report. Others feel that the danger of local invasion, metastasis, hemorrhage and local recurrence with bronchial occlusion outweigh the risk of primary pneumonectomy or lobectomy.[23, 24, 26, 29]

Our attitude toward the treatment of bronchial adenomas has been well summarized in a recent article by McDonald, Moersch and Tinney.[45] In brief, we favor conservative treatment when the adenoma is attached by a narrow pedicle and is movable, when it is situated close to the coryna, and when the general condition of the patient is poor. Lobectomy or pneumonectomy is preferred when the adenoma is situated in a bronchus that cannot be visualized bronchoscopically, when severe secondary pulmonary suppurative disease is present, when there is obvious evidence of peribronchial infiltration (of the adenoma), and when the adenoma has been treated endoscopically and tends to recur. Cylindromas are being treated in a similar manner until more is known about their growth potentialities. We are aware of the advantages of surgical extirpation and are employing this method with increasing frequency.

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A THORACIC SUPPORT

WILLIAM SPICKERS, M.D., F.A.C.S.*†
PATERSON, N. J.

I WISH to describe a simple device to hold patients in a firm, comfortable position during thoracic operations. This support (see Fig. 1) can be made by any hospital carpenter. It consists of a firm horizontal plank of wood, which rests on the table and has a shorter upright piece at right angles fastened at one end of the horizontal piece and a sliding wooden bar with thumbscrews to hold the patient from turning or slipping. The wooden frame is covered with thick quilting (Fig. 2) to protect the patient and to raise somewhat the thoracic cage. All operative procedures are made less difficult by having the patient in a firm, planned position and the operator comfortable.

A piece of adhesive plaster, three or four inches wide, is placed on the operating table, adhesive side up. The thoracic support is placed on this, thereby preventing all movement or displacement of the support.

The patient lies on his well side (Fig. 3), leaning obliquely forward against an upright (Fig. 4). The arms are extended on an arm board, which allows easy access to both arms during the operation, for intravenous therapy, blood transfusion, and blood pressure readings.

It is well to have the patient quite near the edge of the table and a small hard pillow under his head. I always stand in my accustomed position before washing up so that I shall get a proper position of the patient and feel comfortable.

A four-inch wide adhesive plaster is placed over the hip and fastened to the table on each side. The upper leg is extended, lower leg flexed. A small quilt or pillow is placed between the knees and the ankles are tied to the table with bandages or wide tape. After the position is satisfactory, the movable crossbar is pushed against the patient's back and the set screws turned down lightly. This holds the patient firmly in position (Fig. 2).

The anesthetist can then place the patient at any desired angle of reverse Trendelenburg or Trendelenburg without having the patient's position in relation to the table fundamentally altered. I generally use cyclopropane intratracheal anesthesia. Induction is given with the patient on his back, intratracheal tube inserted, and turned on his unaffected side. An intravenous infusion of normal saline solution is started and followed by citrated blood. The remaining arm is used for blood pressure readings. Bone marrow transfusion is given when the veins are poor in both arms and legs.

The support has been used most for thoracoplasty for pulmonary tuberculosis. It is also used for empyema, lobectomy, pneumonectomy, and esophag-

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†Deceased.

Fig. 1.—Thoracic support at usual position near head of table, resting on adhesive tape fixing its position.

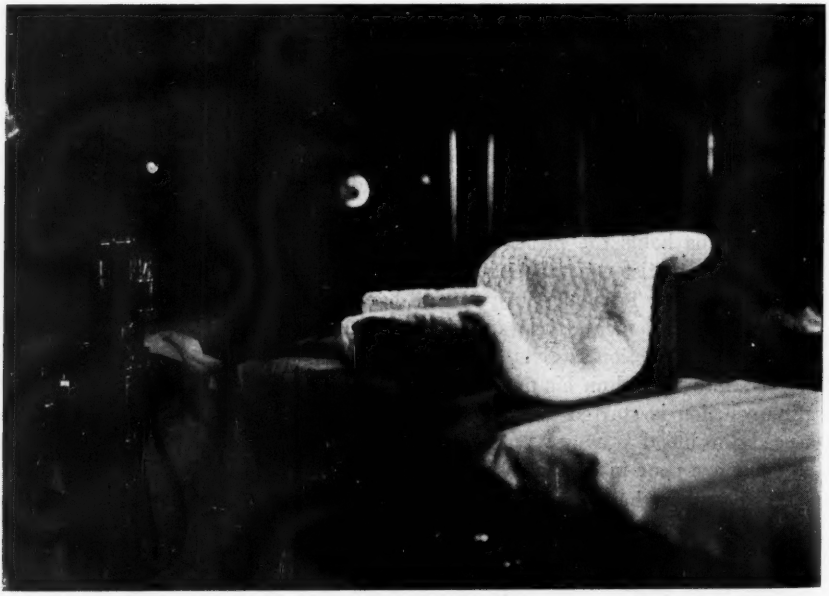

Fig. 2.—Showing heavy quilting and sliding wooden bar.

Fig. 3.—Patient in position, lower leg flexed, upper leg extended and fixed; adhesive plaster over hip is shown, with sliding wooden bar pushed snuggly against patient.

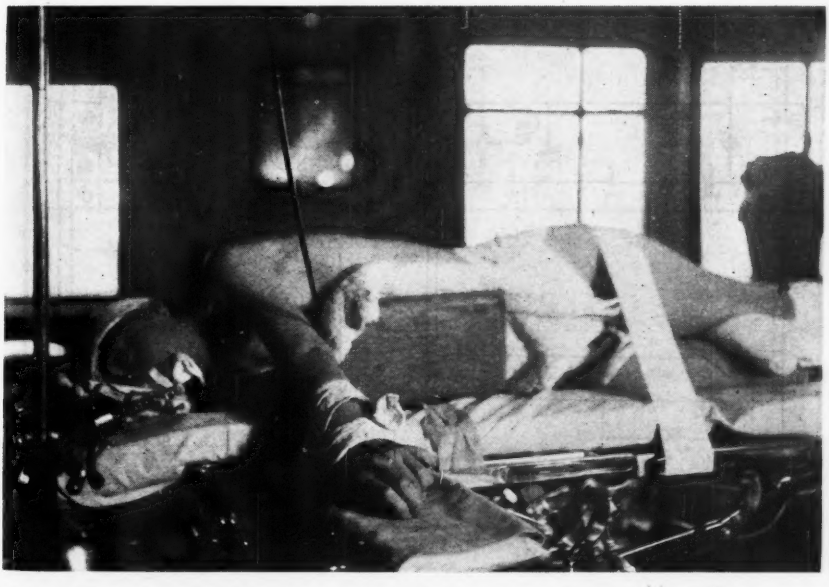

Fig. 4.—Anterior view showing arms extended, blood pressure cuff on arm, and adhesive over hip; bone marrow transfusion is going on in this case.

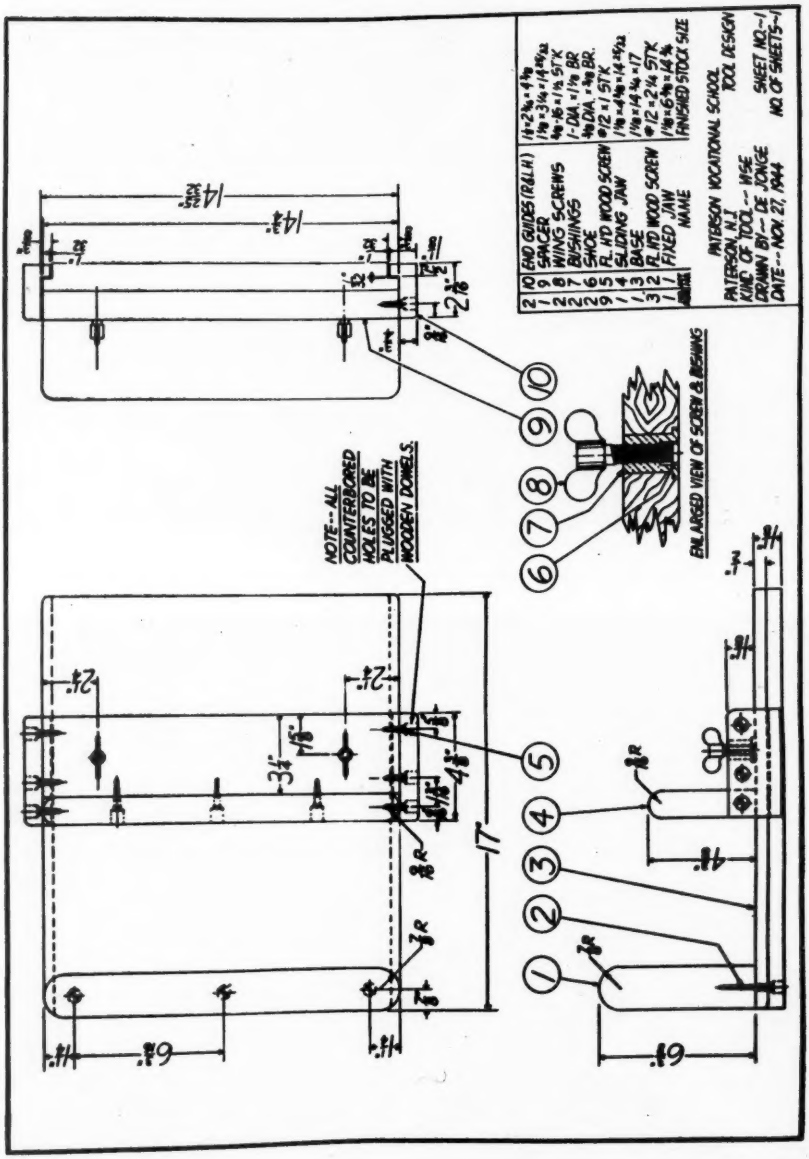
2 10 END GUIDES (R&L H) 1/8 x 2 3/4 x 1 1/8
1 9 SPACER 1/8 x 3 1/4 x 1 1/4
6 8 WING SCREWS 1/4-16 x 1 1/2 ST'K
2 7 BUSHINGS 1" DIA. x 1/2 BR.
2 6 SHOE 3/8 DIA. x 1/2 BR.
9 5 FL. HD WOOD SCREW #12 x 1 ST'K
1 4 SLIDING JAW 1/8 x 4 1/8 x 14 1/2
1 3 BASE 1/8 x 14 1/8 x 17
3 2 FL. HD WOOD SCREW #12 x 2 1/4 ST'K
1 1 FIXED JAW 1/8 x 6 1/8 x 14 1/8
NO. NAME FINISHED STOCK SIZE
PATERSON VOCATIONAL SCHOOL TOOL DESIGN
PATERSON, N.J. SHEET NO.-1
KIND OF TOOL -- WISE NO. OF SHEETS-1
DRAWN BY -- DE JONGE
DATE -- NOV. 27, 1944
NOTE -- ALL COUNTERBORED HOLES TO BE PLUGGED WITH WOODEN DOWELS
ENLARGED VIEW OF SCREW & BUSHING
14 1/2"
2 1/2"
2"
3 1/4"
1 1/8"
4 1/8"
17"
6 1/2"
1/2 R
3/8 R
1/4 R

Fig. 5.—Detailed drawing of thoracic support.

ectomy. It is useful in kidney operations or any other procedure in which the patient has to be firmly held in an oblique or lateral position.

Retractors to hold the scapula away from the chest are being devised so that the retraction can be self-retaining. I propose to do this by having square steel rods fitted into holes in the upright wood section. The retractors will hook under the inner border of the scapula and the handles be fixed to the steel uprights.

The minute drawing (Fig. 5)* is formidable looking but the detailed measurements will be gratefully accepted by the carpenter.

*Made by the Paterson Vocational School.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Twenty-Sixth Annual Meeting

May 29-31, 1946, at Hotel Statler
Detroit, Mich.

Program

Wednesday Morning, May 29, 1946

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **The Management of Thoraco-Abdominal War Injuries**
   Reeve H. Betts
2. **Crushing Injuries of the Chest**
   Ross Robertson
3. **The Pathology of Chronic Hemothorax**
   H. T. Langston and W. M. Tuttle
4. **The Treatment of Organizing Hemothorax**
   W. M. Tuttle, H. T. Langston, and (by invitation) R. T. Crowley
5. **Total Pulmonary Decortication: Evolution and Present Concept**
   P. C. Samson and (by invitation) T. H. Burford

Wednesday Afternoon, May 29, 1946

2:00 P.M.

6. **Thoracic Neoplasms in Navy Personnel**
   William T. Watson
7. **Pulmonary Cysts**
   H. J. Moersch
8. **Report of the Chest Tumor Registry**
   Colonel J. E. Ash (by invitation), Medical Corps
   Director, Army Institute of Pathology
9. **A Report of 180 Lobectomies Performed at Kennedy General Hospital, Chest Surgical Center 1943-1946, With One Death**
   R. H. Meade, Jr., and (by invitation) Earl B. Kay and Felix Hughes
10. **Pulmonary Function After Adolescence Following Pneumonectomy in Childhood**
    André Cournand (by invitation) and Charles W. Lester

5-6 P.M. COCKTAIL PARTY, HOTEL STATLER
(Members and invited guests)

Thursday Morning, May 30, 1946

9:00 A.M.

11. **The Use of Curare in Anesthesia for Thoracic Surgery**
    H. Brodie Stephens and (by invitation) Phyllis Harroun and F. E. Beckett
12. **Surgery of the Thymus in Myasthenia Gravis**
    O. T. Clagett
13. **The Management of Esophageal Injuries and Diseases in an Army Chest Center in This Country**
    E. B. Kay (by invitation)

14. **Esophagectomy and Esophagogastrostomy**
John Garlock

15. **Section of the Vagus Nerve to the Stomach in the Treatment of Peptic Ulcer**
Lester Dragstedt (by invitation)

16. **The Management of Thoracic Surgery in the Hospitals of the Veterans' Administration**
Brian Blades

Thursday Afternoon, May 30, 1946

2:00 P.M. EXECUTIVE SESSION

2:30 P.M. SCIENTIFIC SESSION

Presidential Address: **The Direct Approach to Cardiovascular Disease**
Claude S. Beck

17. **Tetralogy of Fallot, Diagnosis and Indications for Operation**
Helen B. Taussig (by invitation)

18. **The Surgical Treatment of the Tetralogy of Fallot**
Alfred Blalock

19. **The Complications of Surgery of the Patent Ductus Arteriosus**
John Jones

20. **Complete Division of the Patent Ductus Arteriosus**
Robert Gross

21. **Surgery of Coarctation of the Aorta**
Clarence Crafoord

7:00 P.M. COCKTAIL PARTY, HOTEL STATLER

8:00 P.M. BANQUET, HOTEL STATLER

Friday Morning, May 31, 1946

9:00 A.M.

22. **Decortication for Tuberculous Empyema**
Fraser Gurd

23. **Lobectomy and Pneumonectomy in the Treatment of Pulmonary Tuberculosis**
R. H. Sweet

24. **Further Experiences in Pulmonary Resection in the Treatment of Pulmonary Tuberculosis**
R. H. Overholt and (by invitation) N. J. Wilson and L. Langer

25. **Cavernostomy**
E. J. O'Brien and (by invitation) P. V. O'Rourke, F. C. Test, and E. F. Skinner

Friday Afternoon, May 31, 1946

2:00 P.M.

26. **Removal of Foreign Bodies From the Heart and Pericardium**
Dwight Harken (moving picture demonstration)

27. **Cardiac Resuscitation**
Mercier Fanteux (by invitation)

28. **The Management of Traumatic Diaphragmatic Hernia**
Felix Hughes, Earl B. Kay, (by invitation) T. R. Hudson, R. H. Meade, Jr., and Julian Johnson

29. **Thoraco-Gastric Fistula Caused by Surgical Mistreatment of Herniated Stomach**
G. E. Lindskog and (by invitation) E. A. Lawrence

30. **Congenital Eventration of the Diaphragm—Surgical Management, Case Report**
Dewey Bisgard

Notice

The Local Committee on Arrangements is presenting a series of talks and demonstrations by Detroit doctors at Herman Kiefer Hospital on Saturday, June 1, 1946.

9:00 A.M. **Tuberculosis Control**
B. H. Douglas

9:15 A.M. **Vascular Damage Following Pulmonary Disease**
C. C. Birkelo and W. L. Brosius

9:45 A.M. **The Role of Tuberculosis in the Formation of Bronchiectasis Following:**
(1) **Primary Disease**
Edna Jones
(2) **Secondary Disease**
H. S. Willis

10:15 A.M. **Application of Decortication to Civilian Practice**
P. V. O'Rourke and F. C. Test

10:30 A.M. **Thoracic Tumors**
W. A. Hudson

11:00 A.M. **Lung Abscess**
Dewey Dodrill

11:15 A.M. **Clinical Conference: Selection of Cases for Collapse Therapy and Resection**

12:45 P.M. Lunch